A STUDY OF LIPID PROFILE IN NONDIABETICS WITH STROKE

GOVT KILPAUK MEDICAL COLLEGE HOSPITAL, CHENNAI

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

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In Partial Fulfillment of the Regulations

For the Award of the Degree of

M.D.(GENERAL MEDICINE)- BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI

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BONAFIDE CERTIFICATE

This is to certify that the Thesis "**A Study of lipid profile in nondiabetics with stroke"** Conducted in patients of Govt. Kilpauk Medical College Hospital, Chennai is a genuine work done by Dr. S.MYTHILI, Post Graduate Student in the Department of Medicine, Government Kilpauk Medical College under the guidance of PROF. DR. R. SABARATNAVEL M.D., Head of the Department, Department of Medicine, Kilpauk Medical College.

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DECLARATION

I **Dr. S.MYTHILI** solemnly declare that the dissertation titled "**A STUDY OF LIPID PROFILE IN NONDIABETICS WITH STROKE" conducted in patients of Govt Kilpauk medical college Hospital, Chennai"** under the guidance and supervision of **Dr.S.Ushalakshmi, M.D.**,**FMMC., and Dr.G.Balan, M.D.,** Professors of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai. This is submitted to the Tamil Nadu Dr. M.G.R. Medical University Chennai in partial fulfillment of the requirement for the award of MD degree Branch I (General Medicine)

Place: **(Dr.S.MYTHILI)**

Date:

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A study of lipid profile in non diabetics with stroke.

ABSTRACT

Aim: The aim is to study the serum lipid profile in non diabetics with stroke and to determine significant correlation between them.

Objective: The objective is to know about the association between dyslipidemia and non diabetic stroke patients which can help modify our prevention and treatment strategies.

Type of study: cross sectional study

Place of study: Study was conducted on patients of Kilpauk medical college hospital for six months. Patients and controls were tested for fasting lipid profile12 hours after overnight fast.

Participants: Participants were 60 patients of non diabetic stroke and 60 controls. Among the 60 patients 37 were males and 23 were females. In controls there were 37 males and 23 females. Age and sex matched

controls were selected. Stroke patients with infarct or haemorrage in ct brain were included in the study.

Results: In this study total cholesterol, LDL cholesterol and triglycerides were significantly associated with risk of stroke. In this study 56.7% of patients had HDL<40 mg/dl, 41.7% had total cholesterol >200 mg/dl, 65% of them had LDL cholesterol > 100 mg/dl and 43.3 % of patients had VLDL >30 mg/dl.

INTRODUCTION

Stroke or a cerebro-vascular accident is an acute neurological injury which occurs due to vascular pathology and presents as a brain infarction or haemorrhage. Stroke is a medical emergency. The risk factors of stroke have been identified. The modification of risk factors in stroke has brought down both mortality and morbidity of stroke remarkably in the last 30 years.

Dyslipidemia as a major risk factor for stroke is studied for many years. Various studies in different population has shown dyslipidemia is associated with stroke. Dyslipidemia is a correctable risk factor. It has been shown that reduction of total cholesterol, LDL cholesterol, trigycerides, VLDL cholesterol and increasing HDL cholesterol by drugs has decreased the incidence of stroke.

In our study, lipid profile was studied in non-diabetic patients with stroke. Diabetes itself is associated with hyperlipidemia and increased atherosclerosis which makes it an undisputed risk factor for stroke. The atherogenecity of diabetics and non-diabetics are different. So non-diabetic patients were included in the study.

The study is titled as ‘A STUDY OF LIPID PROFILE IN NON-DIABETICS WITH STROKE’

AIM

To study serum lipid profile in non-diabetics with stroke and to determine significant correlation between them.

OBJECTIVE

Knowledge about the association between dyslipidemia and stroke can help to modify our prevention and treatment strategies towards stroke.

The study was conducted on 60 non-diabetic stroke patients and 60 age and sex matched controls who did not have stroke after obtaining informed consent.

This is a cross-sectional study conducted over a period of 6 months in Kilpauk Medical College Hospital.

Detailed history, clinical examination, radiological examination, serum total cholesterol, LDL, VLDL, HDL, triglycerides were estimated by enzymatic method.

INCLUSION CRITERIA

All patients with stroke with hemorrhage or infarct in CT Brain

EXCLUSION CRITERIA

Patients with diabetes mellitus

Patients on drugs for dyslipidemia

Patients on dietary modification for dyslipidemia

Cerebral infarct associated with trauma or tumour

COLLABORATING DEPARTMENTS

Department of Neurology, KMCH

Department of Biochemistry, KMCH

Department of Radiology, KMCH

REVIEW OF LITERATURE

Stroke :-

Stroke-[1] is defined as abrupt onset of neurological deficit, Which is attributable to a focal vascular cause. Stroke is a leading cause of mortality and morbidity throughout the world.

Stroke or Cerebro vascular accident is broadly classified as ischemic and hemorrhagic stroke. TIA occurs when all neurological signs and symptoms of stroke resolve within 24 hours duration regardless of whether imaging evidence of new permanent brain injury is present. If ischemia last more 24 hours but less than 7 days reversible ischemic neurological deficit occur.

Cerebral ischemia is due to reduced blood flow for several second ,if it persists for minutes infarct or brain tissue death occurs. Intracranial hemorrhage due to bleeding into or around the brain may produces stroke. Stroke is a medical emergency.

Dyslipidemia is a modifiable risk factor for stroke. Aggressive management of dyslipidemia decreases the risk of stroke.

Epidemiology of Stroke

WHO collaborative study done in 12 countries showed the incidence of stroke was about 0.2 to 2.5 per 1000 population per year. In India there is not much reliable statistical information. A Random study in urban areas of Vellore showed prevalence rate of hemiplegia in South India as 56.9 per 100000. In developed countries like America stroke causes 2 lakh death per year. About 7,95,000 people have stroke prevalence every year and leading disability and death in America.

Pathophysiology of Stroke :

Stroke is caused by 2 major mechanism ischemia and hemorrhage. Ischemic stroke constitutes 80% of total stroke. Ischemic stroke is due to diminished or absent blood supply to the neurons resulting in deprivation of necessary substrate to neurons. Ischemic damage is rapid because brain has no glucose stores. Glucose is the chief source of energy metabolism and brain is not able to metabolize glucose anaerobically. Intra cerebral hemorrhage constitutes 10 to 15% of all strokes. Bleeding originates from deep penetrating vessels leading to pressure effects and disruption of connecting pathways. Also certain biochemical substances may cause tissue damage by different mechanisms.

Cerebral blood flow :

Normal cerebral blood flow is 50 to 60 ml / 100g / min. When there is ischemia cerebral auto regulatory mechanism compensate the reduced blood flow by vasodilatation, opening of collaterals increased extraction of oxygen and glucose from blood. Cerebral blood flow less than 10ml / 100g/min results in irreversible neuronal injury.

Neuronal injury :-

Microthrombi formation is triggered by ischemia induced activation of destructive vasoactive enzymes which are released by endothelium, leucocytes, platelets and other neuronal cells. Following release of these enzymes mechanical plugging by leucocytes, erythrocytes, platelets and fibrin ensures.

Glutamate and aspartate are neuro transmitters, which when overacts leads to development of hypoxic – ischemic neuronal injury. This is called excitotoxicity which is triggered due to depletion of energy stores. High concentration of glutamate in extra cellular space in energy depleted state results in opening of calcium channels associated with N-methyl D – aspartate and alpha amino 3 hydroxy 5 methyl 4 isoxanol propionate receptors. This persistent membrane depolarisation causes

influx of calcium, sodium and chloride along with efflux of Potasium ions.

The influx of intracellular calcium is responsible for activation of destructive enzymes like Lipase, Proteases and Endonucleases which allow release of cytokines and other substances leading to destruction of cellular integrity Neuronal death occurs by coagulation necrosis and apoptosis. Coagulation necrosis process evolves over 6 to 12 hours. In apoptosis or programmed cell death mechanism begins within 1 hour after ischemic injury.

Cerebral injury is greater with elevated temperature, hypoglycemia, hyperglycemia, hypercoagulable state, hypotension. Good collateral circulation is associated with good outcome. Stroke of short duration and slow onset shows better outcome.

Ischemic Penumbra

Penumbra is the ischemic tissue surrounding the infarct whose dysfunction can be reversed by treatment within critical period of 2-4 hours by reperfusion. Rescue of the ischemic Penumbra is the goal of revascularization therapies. Ischemic penumbra develops within an hour of hypoxia when autoregulatory mechanism fails.

Stroke classification :

Ischemic stroke :

1) Large vessel disease (eg carotid and vertebral stenosis)
2) Small vessel stroke (lacunar stroke)
3) Cardioembolic stroke
4) Thrombosis
5) Undetermined

Primary Hemorrhage:

1. Intracerebral hemorrhage.
2. Subarachnoid hemorrhage.

Causes of stroke

Common causes of stroke

1. Thrombosis

- Lacunar stroke
- Large vessel thrombosis
- Dehydration

2. Embolic occlusion

Cardio embolic

- Atrial fibrillation
- Mural thrombus
- Dilated cardiomyopathy.
- Myocardial infarction
- Valvular lesion
- Mitralstenosis
- Bacterial endocarditis
- Mechanical valve

Paradoxical embolus

- Patent foramen ovale
- Atrial septal defect
- Atrial septal aneurysm
- Echo contrast

Artery to Artery

- Carotid bifurcation
- Aortic Arch
- Arterial dissection

UNCOMMON CAUSES OF STROKE

- Protein c deficiency
- Protein s deficiency
- Antithrombin III deficiency
- Antiphospholipid syndrome
- Factor V leiden mutation
- Malignancy
- B Thalassemia
- Sickle cell anemia
- Polycythemiavera
- Prothrombin G 20210 Mutation
- Homocysteinemia
- Systemic lupus erythematosis
- Nephrotic syndrome
- Disseminated intravascular coagulation

- Oral contraceptives
- Venous Sinus thrombosis
- Vasculitis
- Fibro muscular dysplasia.
- Mitral valve calcification
- Atrial myxoma
- Libmansachs endocarditis
- Marantic endocarditis
- Moya Moya disease
- Eclampsia

Risk factors of stroke

1. Hypertension
2. Diabetes
3. Dyslipidemia
4. Atherosclerosis
5. Atrial fibrillation
6. Coronary artery disease
7. Smoking
8. Age and sex
9. Race
10. Transient Ischemic Attack

Hypertension and Stroke :-

Risk of stroke rises with systolic and diastolic blood pressure. Systolic hypertension is relatively more important than diastolic relative risk with hypertension is 2-5. These is about 38% reduction in relative risk with treatment.

Diabetes :

In diabetes mellitus the relative risk of stroke is double. But tight control of blood sugar has not shown reduction in stroke risk. Dyslipidemia is biochemical abnormally in diabetes associated with high triglycerides, high LDL, low HDL levels. Diabetes also increases atherogenecity.

Dyslipidemia :

Dyslipidemia associated with 1.8 – 2.6 times relative risk of stroke. Stroke prevention by aggressive reduction in cholesterol levels (SPARCL)-[9] trial showed reduction in secondary stroke levels in patients with recent stroke on TIA. The multiple risk factor intervention trial (MRFIT)-[31] with 3,51,000 men with follow up for 6 years showed Increased risk of death from ischemic stroke. Increased with very high cholesterol. Eastern stroke and coronary heart disease study of 70,000

participants showed low cholesterol level associated with low risk of non – hemorrhagic stroke.

Athero sclerosis :-

Thrombosis is late state atherosclerosis, atherosclerosis is the disease of large and medium sized arteries. The most frequent sites are the carotid artery at the carotid sinus, cervical part of the vertebral arteries, basilar artery, bifurcation of middle cerebral artery, Posterior cerebral artery and anterior cerebral artery . The common carotid and vertebral artery at the origin from aorta are frequent sites of atheromatous deposits. Atheromatous plaque narrow lumen of an artery but complete occlusion is due to Thrombosis.

Atherosclerosis leads to hardening of arteries. There is thickening and loss of elasticity of arterial wall. There is focal intimate thickening and lipid accumulation leading to the characteristic plaques and fatty streak.

Atherosclerotic plaques have 3 important components 1. Cells – smooth muscle cells, macrophages, leucocyte 2. Intracellular and extra cellular lipid deposits 37 connective tissue extra cellular matrix, Endothelial damage is a major factor in atherogenesis. Premature atherosclerosis is due to hyper lipoproteinemia and other disorders like

nephritic syndrome, alcoholism. Genetic defects in aproproteins may be associated with accelerated atherosclerosis according to to Breslow J.C. Etal 1992.

Atrial fibrillation

Atrial fibrillation produces emboli which may produce cardio embolic stroke. Atrial fibrillation may occur in rheumatic heart disease like Mitral stenosis. lone Atrial fibrillation increases risk of stroke.

Coronary artery disease :

In Framingham study-[23] it was shown there is a increase in electro cardio graphic changes of LVH in ischemic stroke by 10 folds. Non specific ST and T wave changes also occur.

Smoking :

Framingham study showed increased risk of ischemic stroke in smokers. This risk was also associated significantly with the number of cigarettes Smoked. In male 40% increased incidence of stroke and in female 60% increase in stroke noted.

Age and Sex :

Increasing incidence of stroke with age was shown by Abraham and Daniel et al in 1972. Risk is almost double at about 70 years when compared to age group of 30 – 39 years. Maximum incidence of stroke was shown at 80 years by Agarwal -[21] Etal al. Bansal et al showed a male to female ratio of 3.2:1 in ischemic stroke.

Race:

Stroke is more common in Afro-Caribbean followed by Asians. Stroke is less common in Europeans.

Transient ischemic Attack :

Transient ischemic attacks increase the risk of stroke. There is remarkable increase of about 5% risk of stroke in each year.

Clinical features of stroke

a. Completed stroke

Completed stroke is rapid onset, and persistence of neurological deficit with no progression of neurological deficit more than 96 hours.

b. Evolving stroke

Evolving stroke is characterized by gradual development of neurological deficit. Evolving stroke is due to progression or fluctuation which might be because of propagating emboli which migrates,lyses and disappears and caused by recurrent artery to artery embolization or changes in collateral flow.

c. Transient ischemic attack

In transient ishemic attack the neurological deficit has occurred focally and recovers completely within 24 hours.

d. Reversible ischemic neurological deficit

In RIND, neurological deficit occurs and completely recovers in 1-3 week duration. Signs of ischemia longer than 24 hours and within 7 days constitute reversible ischemic neurological deficit.

1.Athero-thrombotic stroke

Ischemic stroke may be due to thrombosis or embolism. The important cause of athero-thrombotic stroke is atherosclerosis and hypertension. Different pathogenic mechanisms development of

collateral circulation, auto regulation determine the mode of presentation and evolution of stroke.

Onset of stroke during sleep or immediately getting up from sleep is a characteristic feature of thrombotic stroke.

Clinical features depends upon the type of stroke

1. Lacunar infarct

Lacunar infarct is characterized by tiny infarcts in the deeper portion of cerebral hemisphere due to microatheroma or lipohyalinosis of the deep penetrating vessels of internal capsule,thalamus,basal ganglia,para median region of brain stem. It constitutes 10-15% of stroke. Mortality is rare and recovery is common.

2. Embolic infarct

In embolic stroke the source of emboli is mostly cardiac. The emboli consists of fragments of mural thrombus and platelet aggregates. The most common site of embolic occlusion causing infarction is in the middle cerebral artery followed by posterior cerebral artery. Occlusion of anterior cerebral artery is rare. Embolic occlusion occurs very rapidly during any time of the day or night.

The neurological deficit due to emboli may also disappear rapidly due to fragmentation of embolus causing evanescent stroke.

Embolic stroke can become hemorrhagic causing a hemorrhagic infarct. Hemorrhagic infarct is an ischemic infarct resulting due to bleeding within the necrotized cerebral tissue. Hemorrhagic transformation happens when ischemic tissue is reperfused following lysis of emboli spontaneously and blood flow to the ischemic area re-occurs. Hemorrhagic transformation may occur due to persistent occlusion of parent artery proximally.

3. Hemorrhagic stroke

Hemorrhagic stroke may be due to hemorrhage from deep penetrating vessels causing injury to brain tissue. It constitutes 10-15% of all strokes. It is of two types:

- sub-arachnoid
- intra-parenchymal

Hypertension, coagulation abnormalities are high risk for hemorrhagic stroke.

Stroke may present in one of the following ways.

- Stroke in evolution
- Intermittent progression extending over several hours or days
- Completed stroke
- Slow stroke that develops over a period of weeks due to chronic hypo-perfusion.

Symptoms of stroke

- Stroke present as
- Paralysis
- Paresis
- Sensory dysfunction
- Altered sensorium
- Language disturbance
- Higher mental function disturbance
- Cranial nerve dysfunction
- Gait dysfunction
- Headache
- Cerebellar signs
- Seizures

Differential diagnosis of stroke and TIA:

- Metastatic cerebral tumours
- Primary cerebral tumours
- Cerebral abscess
- Todd's paresis
- Sub-dural hematoma
- Peripheral nerve lesions (vascular/compressive)
- Demyelination
- Hypoglycemia
- Encephalitis
- Migrainous aura
- Meniere's disease
- Conversion disorder

Investigations for stroke patients

Basic investigations are done to find out the cause of stroke

Biochemical test

1. Fasting blood sugar
2. Lipid Profile
3. Blood urea
4. Creatinine
5. Electrolytes

Hematological Investigations

1. Complete blood count
2. Prothrombin time.
3. Activated Partial Thromboplastin Time.
4. Peripheral Smear
5. Hematocrit

Urine examination for Protein, sugar, deposits

Special Investigation :

Radio graphic imaging studies

- CT scan (computerized tomography)
- Magnetic resonance imaging
- Doppler
- Ultra sound
- Digital subtraction angiography.

Selected investigations like Hemoglobin electrophoresis, Bone marrow, aspiration, ANA, antithrombin III, Lupus anticoagulant, blood culture in specific cases.

Computerized tomography :

CT Scan may show hyperacute infarct, subacute infarct and chronic infarct. Abnormal perfusion of brain tissue can be made out by injection of contrast media and taking CT scan. This perfusion scan is an useful guide in the treatment of ischemic stroke.

Hyperacute infarcts (<12 hours)

Signs of early infarct can be identified like hyper attenuating artery called as dense MCA sign, insular ribbon sign – gray – white interface

loss along with lateral insular effacement of gray white junction along cortex and lentiform nucleus obscuration.

Sub acute infarct :

After 24 – 48 hours fairly many large vessel infarct are visible on CT scan as wedge shaped areas with diminished attenuation. This infarct involves gray and white matter with vascular distribution mass effect may be present

Chronic infarct :

Well defined focal encephalomalacic areas may be seen on CT Scan. The ipsilaterial ventricle enlarges and adjacent sulci is prominent. Enhancement may disappear over weeks. Infarct may not be visible on CT Scan if it is lacunar infarcts or brainstem infarctsor if the scan is done early. CT Scan done after 2 to 3 weeks may under estimate the size of lesion or may not pick up the lesion due to fogging effect.

MRI Scan :-

In MRI ischemia becomes visible earlier than CT – scan also shows ischemic, necrosis, gliosis. Angiography delineates blood flow and vascular lesions. Can show atheromatous plaques in carotid and vertebral system.MRI is more sensitive compared to CT in finding stroke afftecting brain stem and cerebellum.

MRI can distinguish hemorrhagic from ischemic stroke even after several weeks. The location of hemorrhagic lesion may indicate vascular malformations, saccular aneurysm or myleiod angiopathy. In MRI deep and tiny lacunar infract indicate small vessel disease and a peripheral infarct may indicate embolic etiology.

Digital subtraction angiography :

Used for visualising cervical and basal intracranial arteries and collaterals. Ultrasound and Doppler shows atheromatous plaque and stenosis of large vessels especially carotid arteries.

Duplex ultrasound

Extra cranial arterial disease can be detected non-invasively by duplex ultrasound. It may show atherosclerotic thromboembolic disease in major arteries like carotid.

Electroencephalography (EEG)

Limited value in indicating infarction or differentiation from hemorrhage.

Complications of acute stroke

a. Chest infection

Chest infection can be prevented by nursing the patient in semi-erect position. Aspiration can be avoided by giving nasogastric feeding and avoiding food through mouth. Chest infection can be treated by antibiotics and chest physiotherapy.

b. Epileptic seizures

Adequate cerebral oxygenation has to be maintained and metabolic disturbance has to avoided. Epileptic seizures are treated with anti-convulsants.

c. Deep vein thrombosis/pulmonary embolism

Deep vein thrombosis is prevented by adequate hydration, anti-embolism stockings, early mobilization of patients. Heparin can be used for high risk patients. Treatment is by using anti-coagulants after excluding hemorrhagic stroke.

d. Pressure sores

Pressure sores are prevented by frequent turning of patients, monitoring pressure areas and by avoiding urinary damage to skin. Pressure relieving mattress can be used.

e. Urinary infection

Catherization can be avoided if possible. Urinary tract infection can be treated with antibiotics

f. Constipation

Constipation is prevented and treated by appropriate aperients

g. Painful shoulder

Painful shoulder is avoided by proper shoulder and arm support. Physiotherapy helps in relieving the pain. Local steroid injections are also used.

h. Depression and anxiety

Proper counseling has to be given to the patient to patient regarding stroke. Depression is treated with anti-depressants.

Prognosis of stroke:-

Prognosis depends on the age of the patient. Abraham & Daniel in 1972 showed increasing incidence of stroke with age. Male is about 5 times prone for stroke according to Agarwal-[21] et al 1976. Comorbitidities like hypertension, diabetes atherosclerosis, hyperlipidemia, increase the risk of stroke.

Large infarcts with brain swelling, tentorial herniation, displacement of central structure have worst prognosis. Coma at onset of stroke is poor prognostic sign. Reoccurrence of stroke influences the prognosis. Recovery in embolic stroke is better than thrombotic stroke.

TREATMENT OF STROKE

Ischemic Stroke :-

Cerebral circulation has to maintained by horizontal position of the patient, by maintaining systemic circulation and blood pressure. Initially cerebral edema both cellular and vasogenic edema may occur. cerebral edema is managed by Dexamethaxone, Mannitol, loop diuretics in low doses and oral glycerol. Cerebral vasodilatation is harmful. Intravenous thrombolysis can be done within 3 hours of onset of ischemic stroke in selected group of patient.

Indications of thrombolysis in stroke

1. Clinical diagnosis of stroke
2. Duration of onset of symptoms to drug administration is less than 3 hours.
3. CT-Scan brain shows no hemorrhage or edema of >1/3 of the MCA territory.

4. Age more than 18 years.
5. Consent by the patient or by the surrogate.

Contraindications :-

- Platelet < 1 lakh, hematocrit <25%
- Glucose < 50mg/dl or > 400 mg/dl
- Sustained Bp > 185 / 110 despite treatments.
- Use of heparin within 48 hours and PTT prolongation or INR elevated
- Rapid improvement of symptoms
- Prior stroke or head injury within 3 month.
- Any prior intracranial hemorrhage
- Minor stroke symptoms.
- Any major surgery in the preceding 14 days
- Gastrointestinal bleeding in preceding 21 days.
- Coma or stupor
- Recent Myocardial infarction :

Administration of recombinant tissue Plasminogen :-

- Recombinant tissue plasminogen is administered at the dose of 0.9 mg / kg intravenously (maximum 90mg) iv as 10% total dose by bolus followed by the remaining dose over 1 hour.
- Frequent blood pressure monitoring.
- For 24 hours no other antithrombotic treatment.

  If there is decline in neurological status and uncontrolled blood pressure, stop infusion, give cryoprecipitate and emergency reimaging of brain has to be done.

Antiplatelet drugs like aspirin are useful in preventing thrombotic and embolic strokes. In embolic infarcts prevention of cerebral embolism by long term anticoagulant use and treatment of underlying source of embolus is needed.

Rehabilitation of stroke patient :-

Rehabilitation of the stroke includes physical, occupational and speech therapy. Education of parents and patients regarding neurological deficit, preventing complications of immobity and encouragement for overcoming the deficit.

LIPID METABOLISM

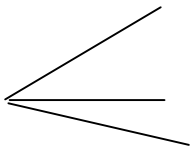
Lipids the lipids are heterogeneous group of compounds comprising of oils, steroids, fats, waxes and related compounds. They are insoluble in water and soluble in non-polar solvents. A Classified as simple lipids and complex lipids.

Simple lipids :

- Fats
- Waxes

Complex lipids

- Phospholipids
- Glycospingo lipids
- Triglycerides
- Sulfolipids and aminolipids
- **Precursor and derived lipids**
- Fatty acids
- Glycerol
- Steroids
- Fatty aldehydes
- Ketone bodies



Cholesterol

Steroids hormones

Vitamin D

Uses of lipids

1) Lipids form structure of cell membrane and is an integral part of cell membrane.
2) Structure of sex hormones.
3) Good source of energy can be used immediately. Caloric value of fat is 9 kilocalories / 1 gm.
4) Act as electrical insulator allowing rapid propagation of depolarization waves in the myelinated nerves.
5) Act as thermal blankets because their presence in subcutaneous tissue protects the body against heat loss.

Fatty acids :

Derived from glycerol and cholesterol. They have even number of carbon atoms and straight chain derivatives. Fatty acids classified as saturated or un saturated fatty acids fatty acids provide for energy requirements.

Unsaturated fatty acids are subdivided as

- Mono unsaturated fatty acids Example Monoenoic acids,
- Polyunsaturated fatty acids (eg. Polyenic acids)
- Eicosinoids (eg. Prostaglandins, thromboxane)

Cholesterol :

Most important sterol.Cholesterol precursor of bile acids, steroid hormones and vitamin D. it is a stable crystalline white substance insoluble in water but soluble in chloroform, ether, alcohol. It is found in high amounts in nervous tissue, skin, liver, intestine and endocrine glands.

Triglycerides :-

Also called as triacyl glycerols Triglycerides are esters of alcohol glycerol and fatty acids eg. monoacyl glycerol, diacyl glycerol and triacyl glycerol.

Lipoprotein coats aggregates of triglyceride (80%) phospholipids 7% and cholesterol (9%) to form chylomicron particles.

Phospholipids – complex lipids with phosphate and nitrogenous base. Eg. Lecithin and sphingomyelin.

Lipoprotein Complex :

Lipids in plasma are in the form of lipoprotein complexes. Complex of lipid and protein makes it soluble and travel in blood stream as lipoprotein complexes.

Lipoprotein in Plasma

Lipoprotein	Major core Lipids	Major Apoproteins
Chylomicrons	Dietary triglyceride	B-48, C, E
HDL	Cholesterol	A1, A-II
LDL	Cholesterol	B-100
VLDL	Endogenous triglyceride	B-100, C, E
Remnants	Triglyceride, Cholesterol	B-100, E

Tiselius et all showed the existence of two lipoprotein classes alpha & Beta lipoprotein electrophoresis. Dangerfield et al identified prebeta lipoprotein by zonal electrophoresis

Characteristic features and structure of lipoprotein

	Chylomicrons	LDL	HDL	VLDL	IDL
Electrophoresis	Origin	Beta	Alpha	Pre-beta	Broad-beta
Diameter	90-1000 nm	20-25nm	20-25 nm	30-90 nm	
Principal core lipid	Exogenous triglyceride	Cholesterol Triglyceride	Cholesterol esters Phospholipids	Triglyceride Cholesterol esters	25-35 nm Triglyceride Cholester-ol esters
Effect on Atheroma	Nil	3+	Protects	1+	2+
Major apoprotein	AI, AII, B-48, CII, CIII, E	B-100	A_1 and A_{II}	B-100, CII, CIII, E	B-100, E
Dietary and Drug treatment	Ineffective	Resins Fibrates, nicotonic and Probucol	Resins Nicotinic acid, fish oils, fibrates probucol	Fibrate Nicotinic acid, fish oils	Fibrate Nicotinic acid

Apolipoprotein :

Apolipoproteinus are genetically determined components of lipoproteins which provide structural stability to lipoproteins like solubilising lipids, activating enzymes and initiating receptor rmediated clearance of the lipoproteins.

Classification of Apolipoprotein .

Apoprotein A – AI, AII

Apoprotein B – B_{48}, B_{100}

Apolipoprotein C – C_{II}, $C_{III,}$ C_{IV}

Apolipoprotein E – E_2, $E_{3,}$ E_4

Apoprotein A :

Apoprotein A_I - A_{II} are major apoprotein of HDL and removes excess cholesterol form surface of cells. Apolipoprotein A is demonstrated as independent risk factor for cardiovascular disease development and better predictor of coronary artery disease than HDL.

Apoprotein B :

Apoprotein B_{48} is major structural protein of chylomicrons. Apoprotein B-100 is major structural protein of VLDL and LDL essential for secretion of VLDL from liver and ligand removal of LDL from receptor of LDL. It may be elevated in patients with coronary artery disease.

Apoprotein C :

Found in all lipoproteins. Apoprotein C regulate the active lipoprotein lipase. ApoC Removes chylomicrons and VLDL by liver .Apoprotein C_2 absence causes hypertriglyceridemia. Apoprotein C_3 prevents the catabolism of VLDL and Chylomicrons.

Apoprotein E :-

Apoprotein E is present in VLDL and Chylomicrons. It is required for catabolism of remnants by specific receptors on liver

Lipoprotein Metabolism :-

Lipoprotein system is used to transport lipids in exogenous system triglycerides are converted to chylomicrons rich in cholesterol by Lipoprotein lipase. In the endogenous system VLDL is secreted by the liver and converted to IDL. IDL is converted to LDL which is rich in cholesterol.

Chylomicrons :

Chylomicrons are derived from dietary fats and cholesterol which is absorbed from the intestine. Chylomicrons are secreted in to the lymph travel through thoracic duct then enter the systemic circulation.

Chylomicrons interact with lipoprotein lipase leading to hydrolysis of triglyceride to fatty acid and glycerol. After lipolysis chylomicrons remnant is released back into circulation which is cleared rapidly by liver by recognition of apoprotein E. Newly secreted chylomicrons are rich in apoproteins B48 and A1. Newly secreted chylomicrons acquired apoproteins C & E from HDL Particles.

VLDL Metabolism

VLDL is synthesized by the liver endogenously the main code lipid is triglyceride, apoprotein B_{100}, C , E are present. Metabolism of VLDL and chylomicrons are similar VLDL transports triglyceride to the tissues which is used as fuel in adipose tissues the transported triglyceride may be used for storage. VLDL interacts with lipoprotein lipase, VLDL remnant. This remnant produced is converted to LDL or cleared by liver by identifying apoprotein E.

LDL Metabolism :

Major component of LDL is cholesterol LDL delivers cholesterol to tissue through as specific high affinity LDL receptor which controls uptake of cholesterol by the cells. LDL receptor controls intra cellular synthesis of cholesterol. Through LDL receptor a portion of the LDL particles catabolised by the liver and the remaining LDL remnant is

plasma. Function of LDL is to supply cholesterol to extra hepatic cells like adrenal cortical cells. Lymphocytes muscles cells and renal cells. They have LDL receptor on the surface. So cholesterol is used and is available to the cell for membranes synthesis. Most cholesterol released from extra hepatic tissue is then transported to the liver for excretion.

HDL Metabolism :

HDL is needed for removing cholesterol from Peripheral tissue to the liver and for metabolising VLDL chylomicrons. Liver and intestine secrete nascent HDL it takes of Cholesterol from VLDL and chylomicrons to become HDL_3. Enzyme LCAT -Lecithin cholesterol acyl transferase transforms HDL_3 to HDL_2 . HDL_2 transfers cholesterol to VLDL or cholesterol directly to liver after conversion to HDL_3 by hepatic triglyceride lipase enzyme.

Fredrickson Classification of hyperlipoproteniemia

Phenotype Lipo Protein Elevated	Chylomicron	IIa LDL	II B LDL and VLDL	III Chylomicron and VLDL	IV VLDL	V Chylomicron and VLDL
Triglyceride	↑↑↑	N	↑	↑↑	↑↑	↑↑↑
Cholesterol	↑	↑↑↑	↑↑	↑↑	N/↑	↑↑
LDL – Cholesterol	↓	↑↑↑	↑↑	↓	↓	↓
HDL – Cholesterol	↓↓↓	N / ↓	↓	N	↓↓	↓↓↓
Plasma Appearance	Opalescent	Clear	Clear	Turbid	Turbid	Opalescent
Pancreatitis	+++	0	0	0	0	+++
Xanthoma	Eruptive	Tendon, tuberous	None	palmar Tubero eruptive	None	Eruptive
Peripheral atherosclerosis	0	+	+	++	+/-	+/-
Coronary atherosclerosis	0	+++	+++	+++	+/-	+/-
Genetic Nomenclature	FCS	FH, FDB, ADH, ARH,	FCHL	FDBL	FHTG	FHTG
Molecular defects	LPL and APOCII	LDL receptor APO-B-100, PCSka, LDLRAP, ABC G5, ABC G8		APOE	APOA-V	APO AV and GPIHBPI

ABBREVIATIONS:

ADH – Autosomal dominant Hypercholesterolemia

APO – Apolipoprotein

ARH – Autosomal recessive hypercholesterolemia.

FCHL – Familal Combined hyperlipidemia

FCS – Familial chylomicronemia syndrome

FDB – Familial defective APO B

FDBL – Familial dysbetalipoproteinemia

FH – Familial hyper cholesterolemia.

FGTG – Familial hyper triglyceridemia

LPL – Lipo Protein Lipase

GPL, HBP – Glycosyl Phosphatidylinositol – anchored high density

Lipo protein binding protein.

LDL RAP – LDL receptor associated protein

N – Normal

Primary disorders of Plasma lipoprotein

Familial lipoprotein lipase deficiency type 1

b. Deficiency of lipoprotein lipase or abnormal lipoprotein lipase or apoprotein CII deficiency causing inactive LPL
c. Autosomal recessive, 1/1000000 incidence.
d. Hypertriglyceridemia
e. Chylomicrons elevated
f. VLDL elevated
g. Cholesterol levels also elevated but lesser degree
h. Clinical finding
    i. Eruptive Xanthomas
    ii. Hepato Splenomeghaly
    iii. Pancreatitis.

Familial hypercholesterolemia – Type II A

- Defect in LDL receptor or mutation in ligand of apo – B – 100
- Autosomal dominant , 1/500 incidence
- Elevated LDL and hypercholesterolemia
- Normal triglyceride

- Elevated LDL is due to increased production of LDL from IDL since there is delayed removal of IDL by LDL receptor mediated endocytosis.

Clinical feature

- Coronary artery disease
- Tendon xanthomas

Familial type II hyperlipoproteinemia

- Deficiency in remant clearance due to abnormality in apo E.
- Isoforms E_3 and E_4 which react with E receptor are deficient.
- Autosomal recessive 1/10000 incidence
- Increased chylomicrons
- Increased VLDL remnants.

Clinical feature

- Palmar and tubereruptive xanthomas
- Coronary artery disease
- Peripheral vascular disease

Familial hypertricylglycerolemia type IV

- Over production of VLDL
- Impaired catabolism of VLDL
- Increase intake of carbohydrates, obesity, insulin resistance, alcohol use, estrogen, treatment increase VLDL synthesis, exacerbate the syndrome.
- Plasma triglycerides increases.
- Increased VLDL
- Reduced plasma HDL

Familial hyperalphalipoprotenimia

- Increased concentration of HDL
- Beneficial to health

Hepatic lipase deficiency

- Deficiency of hepatic lipase
- Autosomal recessive < 1/1000000
- VLDL remnants increased
- HDL increased
- Tendon xanthomas and coronary heart disease present.

Familial lecithin cholesterol acyltranferase (LCAT) deficiency

- Absence of LCAT
- Mutation in LCAT gene.
- Low Plasma HDL
- Hypertriglyceridemia
- VLDL abnormal
- Associated with anemia, progressive corneal opacification, progressive renal insufficiency.

Familial lipoproteinen excess

- Lipoprotein is
- Premature coronary artery disease
- Thrombolysis due to fibrinolysis inhibition.

Hypolipoproteinemias

- Defect in loading of lipid in apo B.
- No chylomicrons
- No VLDL
- No LDL
- Triacylglycerols low.
- Intestinal Malabsorption
- Accumulationof tricicylglycerols in intestine and liver.

Familial alpha lipoprotein deficiency

- Tangier disease
- Fish-eye disease

Apo-A-1 deficiency

- Near absence of HDL
- Low LDL level
- Atherosclerosisoccurs.

Hyperlipoprotenemia secondary to other diseases

a) Diabetes
b) Nephrotic syndrome
c) Hypothyroidism.
d) Pancreatitis
e) Biliary obstruction

Management of hyperlipidemia :

The national cholesterol education program was started in 1985 do decrease the prevalence of elevated blood cholesterol levels. In 1987 adult treatment panel guidelines were released for treating the high cholesterol levels.

This programme recommends all individuals over the age of 20 years and children who are at risk due to atherosclerosis in family must be screened for serum cholesterol which can be collected any time. If S. Cholesterol is less than 200 mg/dl then the person has to repeat the test after 5 years and no further evaluation is needed. Patients with S. Cholesterol > 200mg/dl and other risk factors or with S.cholesterol more than 240mg/dl should undergo complete lipid profile on fasting sample.

Desirable. S. Cholesterol <200mg/dl.

Borderline high 200 – 239 mg/dl

High > 240mg/dl

NCEP recommends the following guidelines for LDL levels

Desirable <100 mg/dl

Borderline 100 – 129 mg/dl

High risk >130 mg/dl

High risk patients should be started with dietary therapy. High risk patients as described by NCEP are patients with definite coronary heart disease or two or more cardio vascular risk factors like male, family history of CHD, smoking, hypertension, diabetes, obesity and low HDL

levels. Drug therapy is started for patients who after dietary therapy have LDL > 129mg/dl and high risk status with HDL<40mg/dl.

The aim of therapy is to reduce LDL to less than 100 mg/dl and in high risk patients to less than 70mg/dl.

Dietary therapy

Dietary therapy is the primary step in the management of patients with hyperlipidemia. In some patients dietary management alone can control hyperlipidemia. The diet advised consists of saturated fat <7% of total calories. Monounsaturated fat upto 20% of total calories polyunsaturated fat 10% of total calories carbohydrate content should be 50 – 60% of total calories, protein, content 15% of total calories, cholesterol content must be less than 200mg/dl. Moderate physical activity leading to expenditure of 200 kcal/day is needed.

Drug therapy :

If dietary therapy fails drug therapy has to be started. Different drugs are available for drug therapy.

ATP III LDL Cholesterol goals and threshold for drug therapy and therapeutic lifestyle changes (TLC)

Category	Start TLC	Start drug therapy	LDL – C Goal
Very high risk	Any LDL – C	LDL C ≥ 70 mg/dl	<70 mg/dl
High Risk	≥100 mg/dl	LDL – C ≥, 100 mg/dl	< 100 mg/dl
Moderately high risk	> 130 mg / dl	LDL –C ≥ 130 mg/dl	< 130mg/dl
Moderate risk	≥130 mg/ dl	LDL-C ≥ 160 mg/dl	< 130 mg/dl
Lower risk	≥ 160 mg/dl	LDL –C ≥ 190mg/dl	< 160 mg/dl

The drug used in dyslipidemia are

1. Cholesterol resins – Cholesterol resins used are colestyramine, colestipol, colesevelam.

    - Lower LDL level 15 to 30%
    - Reduce risk or coronary artery disease (new England Journal of Medicine, 1999)
    - May cause hyper triglyceridemia and should not be used alone.
    - Acts by absorption of bile acids in the intestine so there is no reasorption of bile acids in terminal ileum and normal enterohepatic circulation of bile acids is prevented. Then

cholesterol is converted to bite acids leading to decreased cholesterol. This cholesterol stimulates LDL receptor activity leading to decreased levels of LDL in plasma.

- Dose, cholestyramine 4 to 24 gm po in divided doses. Colestipol 2 to 16 gm PO in divided doses.

Drug interaction:

Decrease oral reabsorption of Amiadarone, Statins, Warfarin, digoxin.

Side effects:

Bloating, Nausea, abdominal pain, constipation, flatulence.

Nicotinic acid :-

- Lowers LDL cholesterol by 15%
- Lowers triglyceride levels 20 to 50% .
- HDL cholesterol increased by 35% (Arch internal medicine 1994)
- Acts by inhibiting secretion of LDL in the liver thereby decreasing LDL levels. Also diminishes mobilisation of free fatty acid from the adipose tissue.
- Dose : 1 to 3 gm orally in 2 or 3 divided does with meals.

Side effects :

Headache, nausea, pruritus, bloating, hyperuricemia, hyperglycemia and elevated liver enzymes. Avoided in Gout, Liver disease, uncontrolled diabetes mellitus.

STATINS :

- HMG coAreductase inhibitors
- Lowers LDL cholesterol 30 – 50%
- Act by inhibiting HMG coA reductase the rate limiting step of cholesterol synthesis.
- Statins increase LDL receptor activity in the liver, removes LDL and VLDL remnants from plasma.
- Statins other important pleiotropic actions like improvement of endothelial function, antioxidant properties, immune modulatory action, inhibition of inflammatory responses, increased bioavailability of nitric oxide, and atherosclerotic plaque stabilization.

Dose :

- Atorvostation 10 – 80 mg, orally /day
- Fluvastatin 20 – 80 mg orally / day
- Lovastatin 10-80 mg orally / day
- Rosuvastatin 5- 40 mg orally/ day
- Pravastatin 10-80 mg orally/ day

Side Effects :

Gastrointestinal upsets abdominal pain, diarrhea, bloating, headache, fatigue, rash. Elevation of liver transaminases 2 or 3 times may occur with statin this is dose dependant and is reversible on drug withdrawal. Liver function test to be performed before starting statins.Muscle pain and muscle weakness can occur without creatinine kinase elevation. Muscle pain and weakness is dependant on the dose of the drug, age, body size, renal parameters and use of other medications.

Statins have increased risk of rhabdomyolysis (new England journal of Medicine 1999) when taken with drugs which undergo metabolism by cytochrome P450 like gemfibrozil, erythromycin, clarithromycin, Ketoconazole, cyclosporin.

Ezetimibe :

- Cholesterol absorption inhibitior acts on the brush border of small intensine.
- Decrease LDL by 18% when used as monotherapy (American journal of cardiology 2002)
- Dose 10 mg orally once a day.

Side effects :-

GI symptoms like nausea, vomiting, diarrhea and myalgias.

Fibric acid derivatives

- Lower triglyceride levels 30 to 50%
- Increase HDL levels10 to 35%
- Lower LDL cholesterol level by 5% to 25%
- Acts by increasing lipoprotein lipase activity and increase VLDL clearance

Dose :

- Gemfibrozil 600 mg orally twice before meals
- Fenofibrate 48 to 145 mg orally per day.

Side effects :

Abdominal discomfort, Myalgia, increased incidence of gall stones, pruritus and rash.

Omega 3 Fatty acids :-

- Lowers triglyceride
- Ingredients are Eicosapetaenoic acid and docosahexaenoic acid.

  Dose 1 to 6 gm of Eicosa pentaenoic acid and docosahexaenoic acid.

METHODOLOGY

COLLECTION OF BLOOD SAMPLE

Blood samples were collected from all patients after an overnight fast of minimum 12 hours. Previous day patient was advised to have light fat free diet. Sample collected from cubital fossa. Tourniquet was released just before sample collection to avoid increased serum lipids artefactually. 10ml of blood was drawn in sterile syringes and blood was transferred to dry glass tubes.

PREPARATION OF SERUM

Serum for HDL was separated within two hours of collection. The sample was centrifuged at 5000 rpm for 10 minutes in a centrifuge tube. The clear serum was pipetted out and stored at 4 degree centigrade. Samples were analysed within 24 hours.

SERUM TOTAL CHOLESTEROL ESTIMATION.

Serum total cholesterol is measured by cholesterol peroxidase method. This method has extended stability. Reconstituted reagent is

stored at 2-8 degree centigrade and is stable for 90 days. This method is linear up to 500 mg/dl.

PRINCIPLE

Enzymatic calorimetric method of determination of total cholesterol.

The following reactions

Cholesterol esterase

Cholesterol + water ----------> fatty acid+ cholesterol

Cholesterol esterase

Cholesterol + oxygen ----------> 4-cholesten-3-one+H2O2

Peroxidase

2H2O2+ phenol+4-aminoantipyrine------> red quninone+4H20

The reagent is stable when stored at 2-8 degree centigrade upto expiry date.

The reagent is linear upto the value of 500 mg/dl, if the concentration is greater than 500 mg/dl, the sample has to be diluted with

normal saline and the assay has to be repeated and the result has to be repeated with dilution fraction.

REAGENTS

CHOLESTEROL R1:2x50 ml/4x50ml/4x100ml/2x405ml

Phenol-24mmol/L

Sodium cholate- 0.2mmol/L

Pipes buffer(pH-6.9)-50mmol/L

CHOLESTEROL R2:2x50ml/4x50ml/4x100ml/8x100ml

Cholesterol esterase >200U/L

Peroxidase >1000U/L

Cholesterol oxidase<250U/L

4-aminoantipirine – 0.5mmol/L

CHOLESTEROL STANDARD:1x5ml/1x5ml/1x5ml/2x5ml

Cholesterol standard concentration 200mg/dl

PREPARATION OF WORKING REAGENT

Dissolve reagent R1 and R2 of cholesterol as shown on the label

SAMPLE- Serum

	BLANK	STANDARD	SAMPLE
Working reagent	1000µL	1000µL	1000µL
Standard	-	10µL	-
Sample	-	-	10µL

Mix the contents and incubate at 37 degree centigrade for 5 minutes. Absorbance of the standard and sample to be measured against reagent blank.

CALCULATION

Cholesterol conc(mg/dl)= absorbance of sample/ absorbance of standard x 200

ESTIMATION OF SERUM HDL CHOLESTEROL:

The reagent measures HDL cholesterol in serum/plasma by precipitation method, linear up to 125 mg/dl.

REAGENT COMPOSITION

HDL CHOLESTEROL REAGENT : 4x25ml

Magenesium chloride-1mmol/L

Phosphotungstate-14mmol/L

HDL CHOLESTEROL CONCENTRATION STANDARD-1x5ML

HDL CHOLESTEROL CONCENTRATION :50mg/L

PRINCIPLE

VLDL,LDL,Chylomicrons are precipitated by magnesium and phosphotungstate. High density lipoproteins are concentrated in the supernatant following centrifugation is measured by enzymatic methods.

REAGENT

Reagent is stored at 2-8 degree centigrade and is stable upto expiry date. The reagent is linear upto the value of 125 mg/dl, if the concentration is greater than 125 mg/dl, the sample has to be diluted with normal saline and the assay has to be repeated and the result has to be repeated with dilution fraction.

The reagent can be used readily.

SAMPLE: serum/plasma

PROCEDURE

SAMPLE	300µL
HDL reagent	300µL

Mix both reagent and sample allow it to stand for 10 minutes at room temperature. Remix and centrifuge for 10 minutes at 4000rpm. Separate the clear precipitant within one hour and HDL cholesterol concentration has to be determined.

	BLANK	STANDARD	SAMPLE
Working reagent	1000µL	1000µL	1000µL
Standard (HDL)	-	50µL	-
Sample (HDL Supernatant)	-	-	50µL

Mix the contents and incubate at 37 degree centigrade for 5 minutes. Absorbance of the standard and sample to be measured against reagent blank.

CALCULATION

HDL Cholesterol conc. (mg/dl)= absorbance of sample/ absorbance of standard X NX2

Where 2 is dilution factor of sample

N is the standard concentration

SERUM LDL

The immunological principle together with enzymatic assay of cholesterol is used for estimation of LDL directly.

LDL=Total cholesterol – (HDL Cholesterol + triglyceride/5)

SERUM TRIGLYCERIDE

GPO-PAP methodology is used for measuring triglycerides in serum or plasma.

Reagent is stored at 2-8 degree centigrade and is stable up to expiry date. The reagent is linear up to the value of 1000 mg/dl, if the concentration is greater than 1000 mg/dl, the sample has to be diluted with normal saline and the assay has to be repeated and the result has to be repeated with dilution fraction.

PRINCIPLES

Enzymatic determination of triglycerides is by following reaction

Lipoprotein lipase

Triglyceride + water ----------> glycerol+fatty acid

Glycerol kinase

Glycerol+ATP---------->glycerol-3- phosphate+ADP

Glycerol 3 phosphate oxidase

Glycerol 3 phosphate+ oxygen ----------> dihydroxyl acetone phosphate+H202

POD

H202+4-aminoantipirine+P-chloro phenol -------->red quinoneimine

REAGENT COMPOSITION

TRIGLYCERIDE RI: 3X10ml/5X25ml/5X50ml

Potassium ferrocynate- 10mmol/L

Magnesium salt-17mmol/L

4-aminoantipyrine-0.9mmol/L

P-chlorophenol-5.3mmol/L

Pipes buffer(pH 7)-50mmol/L

Lipoprotein lipase ≥1800 U/L

Glycerol kinase ≥ 450 U/L

Peroxidase ≥ 450 U/L

Glycerol 3 phosphate oxidase ≥ 3500 U/L

TRIGLYCERIDE STANDARD CONCENTRATION – 200mg/dl

SAMPLE- serum/plasma

	BLANK	**STANDARD**	**SAMPLE**
Working reagent	1000µL	1000µL	1000µL
Standard	-	10µL	-
Sample	-	-	10µL

Mix the contents and incubate at 37 degree centigrade for 5 minutes. Absorbance of the standard and sample to be measured against reagent blank.

Triglyceride concentration (mg/dl)= absorbance of sample/ absorbance of standard X 200

VLDL concentration(mg/dl)= triglyceride/5

STATISTICAL ANALYSIS

Group Statistics

	Diagnosis	N	Mean	d. Deviatic	td. Erro Mean
Age	Non Diabetics w	60	58.62	12.209	1.576
	Control	60	58.20	11.725	1.514
BMI	Non Diabetics w	60	4.9113	2.92865	.37809
	Control	60	4.2908	1.83463	.23685

Independent Samples Test

		evene's Test fo uality of Varian		t-test for Equality of Means						
									5% Confidenc Interval of the Difference	
		F	Sig.	t	df	g. (2-taile	Mean ifferenc	td. Erro ifferenc	Lower	Upper
Age	Equal vari assumed	.002	.964	.191	118	.849	.417	2.185	-3.911	4.744
	Equal vari not assum			.191	7.808	.849	.417	2.185	-3.911	4.744
BMI	Equal vari assumed	9.364	.003	1.391	118	.167	.62050	.44615	26299	50399
	Equal vari not assum			1.391	9.127	.167	.62050	.44615	26474	50574

AGE GROUP X DIAGNOSIS

TABLE 1

Age Group * Diagnosis Crosstabulation

			Diagnosis		
			Non Diabetics with stroke	Control	Total
Age Group	< 40 Years	Count	5	5	10
		% within Diagno	8.3%	8.3%	8.3%
	41 - 60 Yea	Count	29	29	58
		% within Diagno	48.3%	48.3%	48.3%
	> 60 Years	Count	26	26	52
		% within Diagno	43.3%	43.3%	43.3%
Total		Count	60	60	120
		% within Diagno	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Squar	.000[a]	2	1.000
Likelihood Ratio	.000	2	1.000
Linear-by-Linear Association	.000	1	1.000
N of Valid Cases	120		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.00.

BMI X DIAGNOSIS

TABLE 2

BMI * Diagnosis Crosstabulation

			Diagnosis		
			Non Diabetics with stroke	Control	Total
BMI	UNDER WEIGHT	Count	1	0	1
		% within Diagnosis	1.7%	.0%	.8%
	NORMAL	Count	28	40	68
		% within Diagnosis	46.7%	66.7%	56.7%
	OVER WEIGHT	Count	30	20	50
		% within Diagnosis	50.0%	33.3%	41.7%
	OBESE	Count	1	0	1
		% within Diagnosis	1.7%	.0%	.8%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Squar	6.118[a]	3	.106
Likelihood Ratio	6.915	3	.075
Linear-by-Linear Association	3.601	1	.058
N of Valid Cases	120		

a. 4 cells (50.0%) have expected count less than 5. T minimum expected count is .50.

Sex X Diagnosis

TABLE 3

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
Sex	Male	Count	37	37	74
		% within Diagnosis	61.7%	61.7%	61.7%
	Female	Count	23	23	46
		% within Diagnosis	38.3%	38.3%	38.3%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.000[b]	1	1.000		
Continuity Correction[a]	.000	1	1.000		
Likelihood Ratio	.000	1	1.000		
Fisher's Exact Test				1.000	.574
Linear-by-Linear Association	.000	1	1.000		
N of Valid Cases	120				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 23. 00.

Smoking X Diagnosis

TABLE 4

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
Smoking	Yes	Count	14	0	14
		% within Diagnosis	23.3%	.0%	11.7%
	No	Count	46	60	106
		% within Diagnosis	76.7%	100.0%	88.3%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	15.849[b]	1	.000		
Continuity Correction[a]	13.666	1	.000		
Likelihood Ratio	21.263	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	15.717	1	.000		
N of Valid Cases	120				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 00.

Hypertension X Diagnosis

TABLE 5

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
Hypertension	Yes	Count	14	0	14
		% within Diagnosis	23.3%	.0%	11.7%
	No	Count	46	60	106
		% within Diagnosis	76.7%	100.0%	88.3%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	15.849[b]	1	.000		
Continuity Correction[a]	13.666	1	.000		
Likelihood Ratio	21.263	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	15.717	1	.000		
N of Valid Cases	120				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 00.

T-TEST

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
T.CHOLESTEROl	Equal variances assumed	77.662	.000	5.463	118	.000	47.567	8.707	30.325	64.809
	Equal variances not assumed			5.463	84.419	.000	47.567	8.707	30.253	64.880
TGL	Equal variances assumed	24.745	.000	2.230	118	.028	13.350	5.985	1.497	25.203
	Equal variances not assumed			2.230	87.069	.028	13.350	5.985	1.454	25.246
HDL	Equal variances assumed	13.005	.000	-1.332	118	.186	-2.117	1.590	-5.264	1.031
	Equal variances not assumed			-1.332	104.021	.186	-2.117	1.590	-5.269	1.035
LDL	Equal variances assumed	57.909	.000	6.209	118	.000	48.9567	7.8852	33.3418	64.5715
	Equal variances not assumed			6.209	85.422	.000	48.9567	7.8852	33.2799	64.6334
VLDL	Equal variances assumed	5.372	.022	1.624	118	.107	6.6383	4.0864	-1.4539	14.7305
	Equal variances not assumed			1.624	61.081	.109	6.6383	4.0864	-1.5327	14.8094
HDL | LDL	Equal variances assumed	3.274	.073	-2.844	118	.005	-.15148	.05326	-.25695	-.04602
	Equal variances not assumed			-2.844	97.487	.005	-.15148	.05326	-.25718	-.04579
TC | HDL	Equal variances assumed	36.058	.000	5.674	118	.000	1.55168	.27346	1.01015	2.09322
	Equal variances not assumed			5.674	77.853	.000	1.55168	.27346	1.00724	2.09613

Mann-Whitney Test

Ranks

	Diagnosis	N	Mean Rank	Sum of Ranks
VLDL	Non Diabetics with st	60	66.28	3976.50
	Control	60	54.73	3283.50
	Total	120		
HDL | LDI	Non Diabetics with st	60	45.01	2700.50
	Control	60	75.99	4559.50
	Total	120		

Test Statistics[a]

	VLDL	HDL | LDL
Mann-Whitney U	1453.500	870.500
Wilcoxon W	3283.500	2700.500
Z	-1.820	-4.880
Asymp. Sig. (2-tailed)	.069	.000

a. Grouping Variable: Diagnosis

T.Cholesterol X Diagnosis

TABLE 6

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
T.CHOLESTERO	< 200	Count	26	54	80
		% within Diagnosi	43.3%	90.0%	66.7%
	200 - 240	Count	10	3	13
		% within Diagnosi	16.7%	5.0%	10.8%
	> 240	Count	24	3	27
		% within Diagnosi	40.0%	5.0%	22.5%
Total		Count	60	60	120
		% within Diagnosi	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	29.903[a]	2	.000
Likelihood Ratio	32.580	2	.000
Linear-by-Linear Association	28.484	1	.000
N of Valid Cases	120		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.50.

TGL X Diagnosis

TABLE 7

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
TGL	< 150	Count	35	52	87
		% within Diagnosis	58.3%	86.7%	72.5%
	150 - 199	Count	16	4	20
		% within Diagnosis	26.7%	6.7%	16.7%
	>_ 200	Count	9	4	13
		% within Diagnosis	15.0%	6.7%	10.8%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	12.445[a]	2	.002
Likelihood Ratio	13.027	2	.001
Linear-by-Linear Association	8.828	1	.003
N of Valid Cases	120		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.50.

HDL X Diagnosis

TABLE 8

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
HDL	< 40	Count	32	22	54
		% within Diagnosis	53.3%	36.7%	45.0%
	>_ 40	Count	28	38	66
		% within Diagnosis	46.7%	63.3%	55.0%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.367[b]	1	.067		
Continuity Correction[a]	2.727	1	.099		
Likelihood Ratio	3.384	1	.066		
Fisher's Exact Test				.098	.049
Linear-by-Linear Association	3.339	1	.068		
N of Valid Cases	120				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 27. 00.

LDL X Diagnosis

TABLE 9

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
LDL	< 100	Count	21	55	76
		% within Diagnosis	35.0%	91.7%	63.3%
	100 - 130	Count	5	0	5
		% within Diagnosis	8.3%	.0%	4.2%
	131 - 160	Count	10	3	13
		% within Diagnosis	16.7%	5.0%	10.8%
	>160	Count	24	2	26
		% within Diagnosis	40.0%	3.3%	21.7%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	42.595[a]	3	.000
Likelihood Ratio	48.613	3	.000
Linear-by-Linear Association	37.318	1	.000
N of Valid Cases	120		

a. 2 cells (25.0%) have expected count less than 5. The minimum expected count is 2.50.

HDL | LDL X Diagnosis

TABLE 10

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
HDL | LDL	< 0.39	Count	43	10	53
		% within Diagnosis	71.7%	16.7%	44.2%
	>_ 0.4	Count	17	50	67
		% within Diagnosis	28.3%	83.3%	55.8%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	36.801[b]	1	.000		
Continuity Correction[a]	34.604	1	.000		
Likelihood Ratio	39.122	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	36.494	1	.000		
N of Valid Cases	120				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 26. 50.

TC | HDL MALE X Diagnosis

TABLE 11

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
TC | HDL MALE	< 4.4	Count	16	32	48
		% within Diagnosis	43.2%	86.5%	64.9%
	> 4.5	Count	21	5	26
		% within Diagnosis	56.8%	13.5%	35.1%
Total		Count	37	37	74
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	15.179[b]	1	.000		
Continuity Correction[a]	13.341	1	.000		
Likelihood Ratio	16.024	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	14.974	1	.000		
N of Valid Cases	74				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 13.00.

TC | HDL FEMALE X Diagnosis

TABLE 12

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
TC | HDL FEMALE	< 3.9	Count	5	15	20
		% within Diagnosis	21.7%	65.2%	43.5%
	>_ 4.0	Count	18	8	26
		% within Diagnosis	78.3%	34.8%	56.5%
Total		Count	23	23	46
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	8.846[b]	1	.003		
Continuity Correction[a]	7.165	1	.007		
Likelihood Ratio	9.180	1	.002		
Fisher's Exact Test				.007	.003
Linear-by-Linear Association	8.654	1	.003		
N of Valid Cases	46				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected coun
00.

VLDL X Diagnosis

TABLE 13

Crosstab

			Diagnosis		
			Non Diabetics with stroke	Control	Total
VLDL	< 30	Count	34	52	86
		% within Diagnosis	56.7%	86.7%	71.7%
	>_ 30	Count	26	8	34
		% within Diagnosis	43.3%	13.3%	28.3%
Total		Count	60	60	120
		% within Diagnosis	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	13.297[b]	1	.000		
Continuity Correction[a]	11.860	1	.001		
Likelihood Ratio	13.829	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	13.186	1	.000		
N of Valid Cases	120				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected cour
00.

DISCUSSION ABOUT THE TABLES

TABLE : 1

The data on group statistics shows the association between non-diabetics with stroke and their control with age. The mean age for non-diabetics with stroke is 58.62 and the standard deviation is 12.209.

In controls the mean age is 58.20 and the standard deviation is 11.725. maximum number of patients occur in the age group of 41-60 years in both controls and patients is 48.3%

Significance is 1.0 which in not statistically significant.

TABLE : 2

The data on group statistics shows the association between non-diabetics with stroke and their control with BMI. The mean BMI of non-diabetics with stroke is 24.911 and the standard deviation is 2.92865.

In controls the mean BMI is 24.91 and the standard deviation is 1.83. Maximum patients in the non- diabetic stroke group fall in overweight category and is 50%. In control group the maximum number of patients fall in normal category and is 66.7% significance is 0.106 which is not significant statistically.

TABLE : 3

The data shows association between non-diabetics with stroke and their control with sex. In both patients and controls male constituted 61.7% and female constituted 38.3%. the significance was 1.0 is not statistically significant.

TABLE : 4

The data shows association between non-diabetics with stroke and their control with smoking. In non-diabetes with stroke smokers constituted 23.3% and control group had no smokers. The significance was 0.000 (P< 0.001) and is highly significant.

TABLE : 5

The data shows association of non-diabetics with stroke and their control with hypertension. In patients 23.3% were associated with hypertension and in control group had no hypertension. The significance was 0.000 (P<0.0001) and is highly significant.

TABLE : 6

The data shows associations of non-diabetics with stroke and their controls with total cholesterol 43.3% of stroke patients had normal total cholesterol value and 56.7% had high total cholesterol values. In control

group 90% had normal total cholesterol values and 10% had high T. cholesterol values. The significance calculated was 0.000 (P<0.001) which is highly significant.

TABLE : 7

The data shows association of non-diabetics with stroke and their controls with triglycerides 58.3% of non – diabetics with stroke had normal triglycerides and 41.7% of non – diabetics had elevated triglycerides. In control group 86.7% had normal triglycerides and 13.3% had elevated triglycerides, the significance calculated was 0.002 (P < 0.05) which is significant.

TABLE : 8

The data shows association of non- diabetics with stroke and the controls to high density lipoproteins. Majority of the patients with stroke. 53.3 % had low HDL Values in contrast to controls. Where 63.3% had high HDL values. The significance calculated was 0.067 which is not significant.

TABLE : 9

The data shows association of non-diabetics with stroke and the controls to low density lipoproteins. Only 35% of the patients had normal

LDL cholesterol values, 65% of the patients had high LDL cholesterol values. In control group 91.7% had normal LDL cholesterol values. The remarking 8.3% had high LDL values. The significant calculated was 0.000 (P>0.001) is highly significant.

TABLE : 10

The data shows association of non-diabetics with stroke and controls with HDL / LDL ratio. In non-diabetics with stroke majority 71.7% had HDL/LDL ratio <0.39 only 28.3% of stroke patients had normal HDL / LDL ratio more than 0.4. In control group 83.3% had HDL / LDL ratio >0.4 which is normal. Remaining controls 16.7% had low HDL / LDL values. The significance calculated was 0.000 (P<0.001) which is highly significant.

TABLE : 11

The data shows association of non – diabetics with stroke and controls with TC / HDL ratio in male. In 56.8% of non-diabetics with stroke had TC / HDL ratio $\geq$ 4.5. The TC / HDL in non-diabetics with stroke < 4.4 were 43.2%. In control group 86.5% had TC / HDL ratio <4.4 which is normal. In control group 13.5% had TC / HDL ratio $\geq$ 4.5. On calculation significance was 0.000 is highly significant (P<0.001).

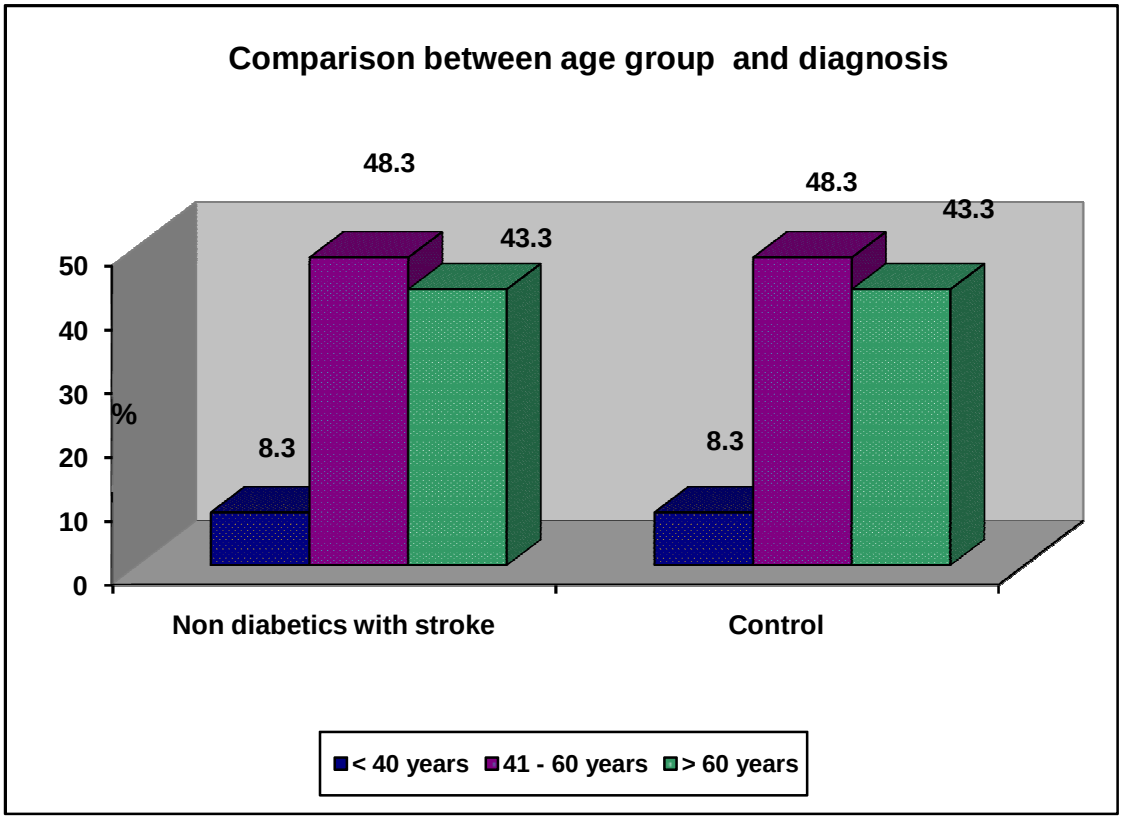
TABLE : 12

The data shows association of non – diabetics with stroke and control with TC / HDL ratio in female. In 78.3% had TC / HDL ratio $\geq$ 4.0 only 21.74% had TC / HDL < 3.9 which is normal. In control group 65.2% had TC / HDL ratio < 3.9. 34.8% of the control had TC / HDL ratio $\geq$ 4.0. The statistically significance calculated was 0.003 which is significant P value < 0.05.

TABLE : 13

The data shows association of non –diabetics with stroke and control with VLDL cholesterol 56.7% of non – diabetics VLDL < 30. In 43.3% of patient showed VLDL > 30. In control group 86.7% had VLDL < 30 and 13.3 % of control had VLDL $\geq$ 30. Significance calculated was 0.000 P (<0.001) which is highly significant.

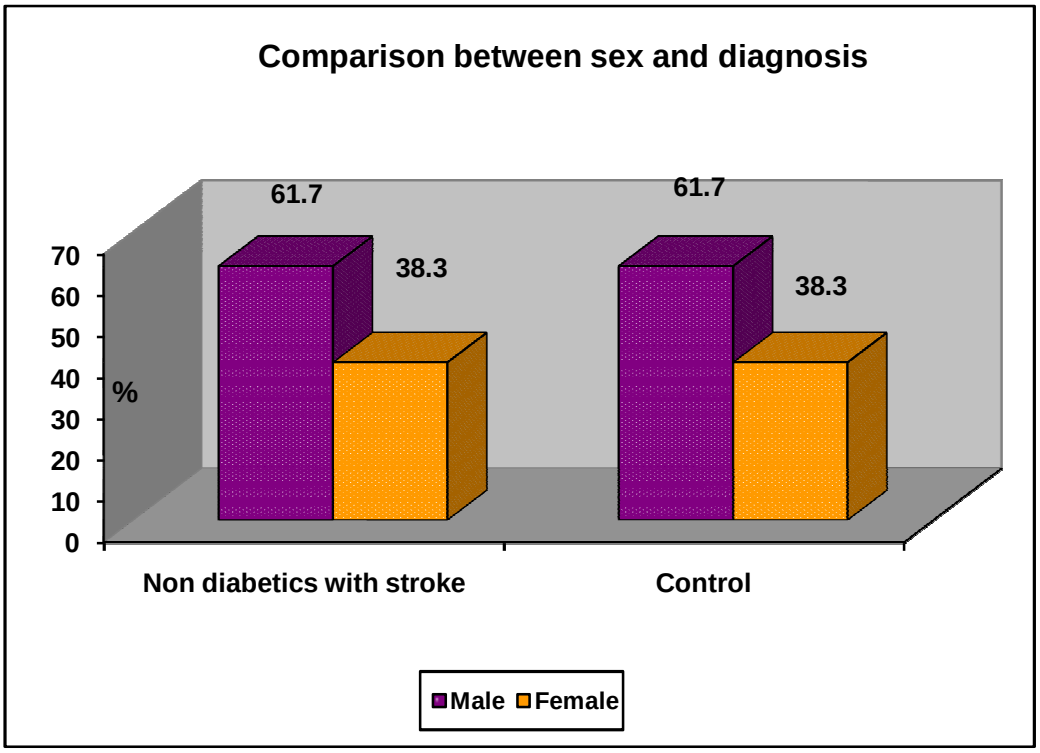
AGE X DIAGNOSIS



In both control groups and people with non-diabetic stroke<40 years is 8.3%. In 41-60 years its 48.3% and > 60 years is 43.3%. Maximum number of patients in 41-60 years group is 48.3%.

SEX X DIAGNOSIS

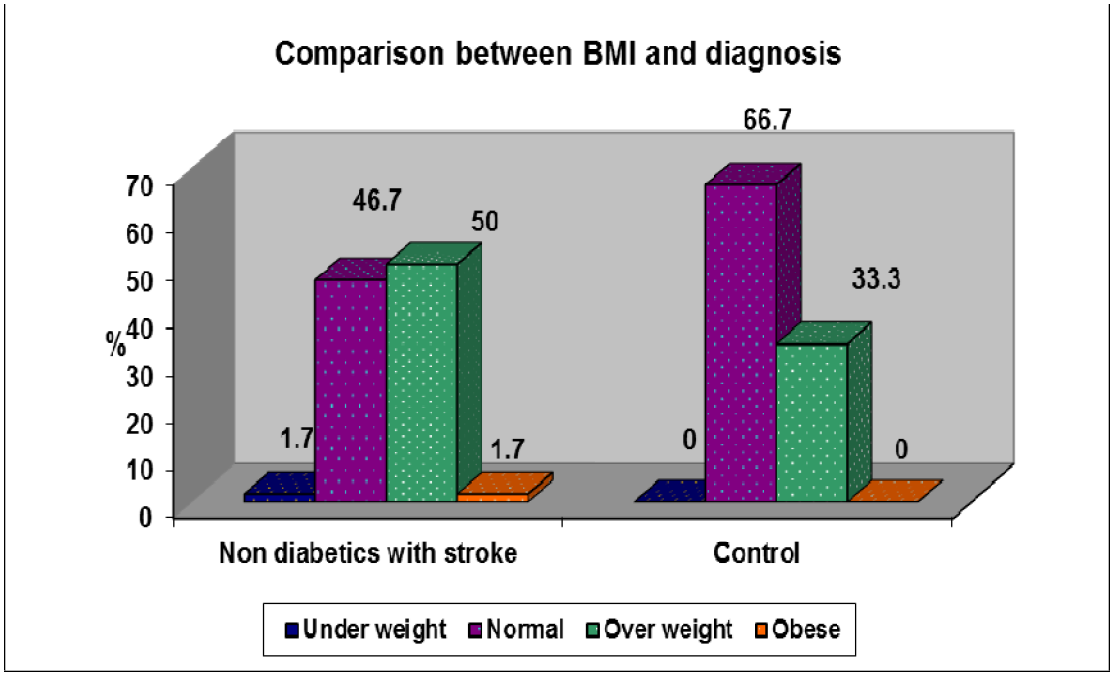
Gender	Non diabetics with stroke	Control
Male	61.7	61.7
Female	38.3	38.3



Graph 2 shows sex-wise distribution. In both groups, 61.7% of the patients were males and 38.3 % were females. Male to female ratio is 1.61:1.

BMI X DIAGNOSIS

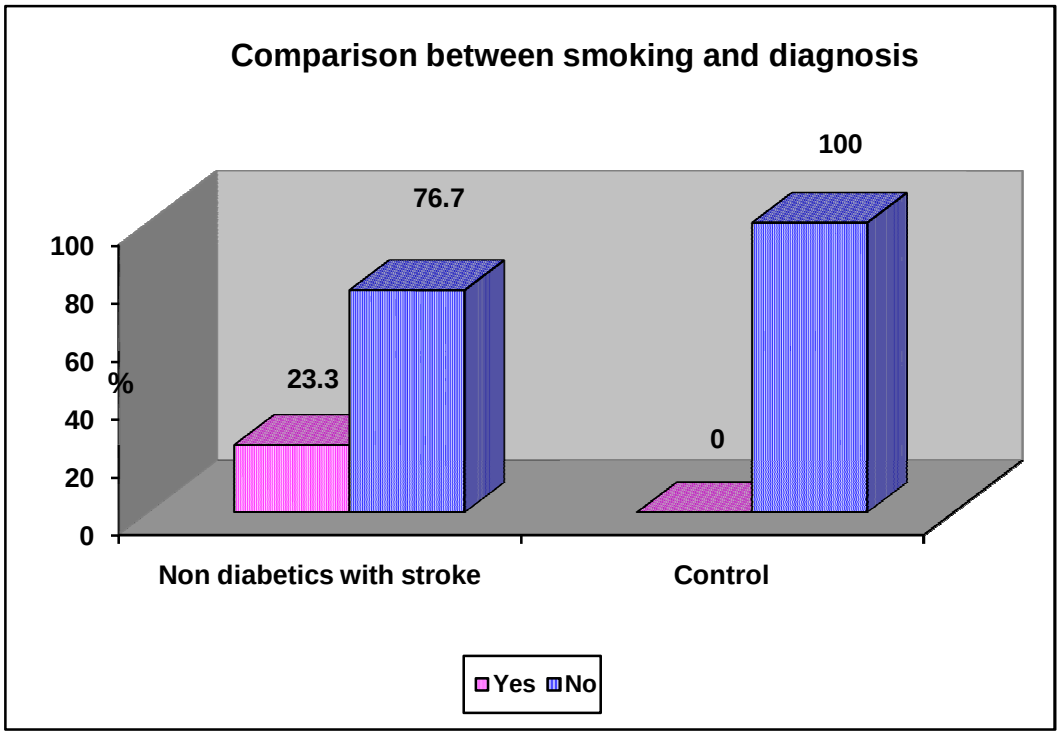
BMI	Non diabetics with stroke	Control
Under weight	1.7	0
Normal	46.7	66.7
Over weight	50	33.3
Obese	1.7	0



Graph 3 shows comparison between BMI in non-diabetics with stroke and control. In non-diabetics, 1.7% was under-nourished, 46.7% was normal, 50% overweight, 1.7% obese. Maximum number of patients- 50% was over-weight. In control 66.7% was normal and 33.3% was over-weight.

SMOKING X DIAGNOSIS

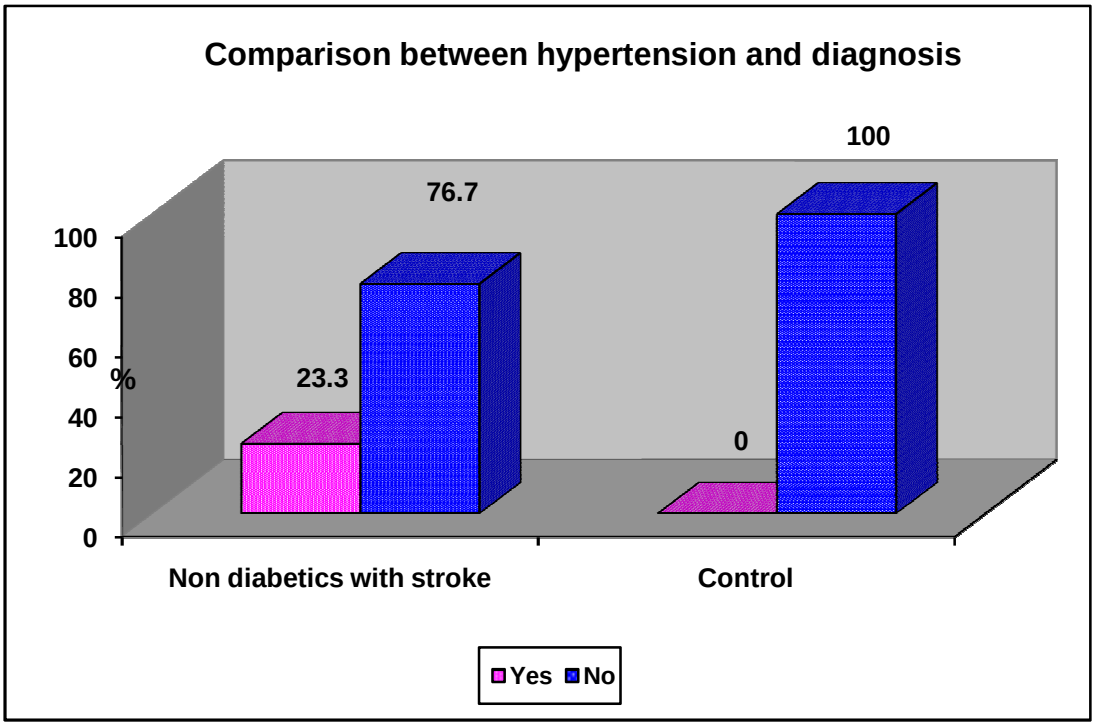
SMOKING	Non diabetics with stroke	Control
Yes	23.3	0
No	76.7	100



In non-diabetics with stroke, 23.3 % were smokers and 76.7% were non-smokers. In control group, 100% were smokers

HYPERTENSION X DIAGNOSIS

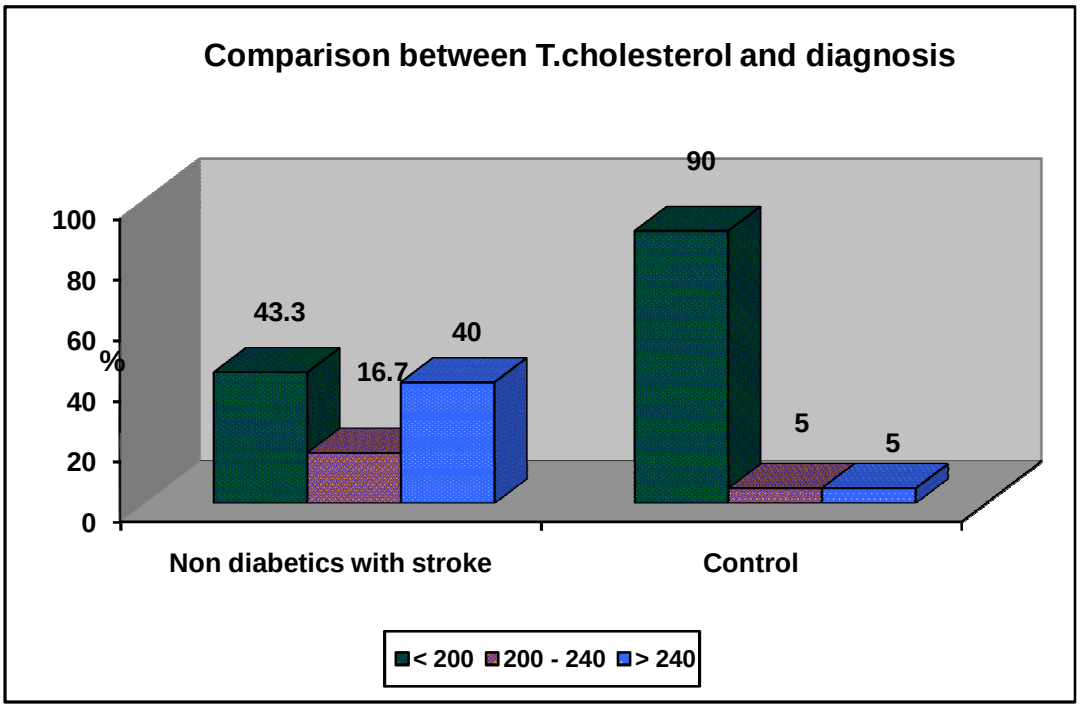
Hypertension	Non diabetics with stroke	Control
Yes	23.3	0
No	76.7	100



Graph 5 shows, 23.3% of non-diabetics with stroke had hypertension, 76.7% of the same group were normo-tensives. All controls 100% were normo-tensives.

TOTAL CHOLESTEROL X DIAGNOSIS

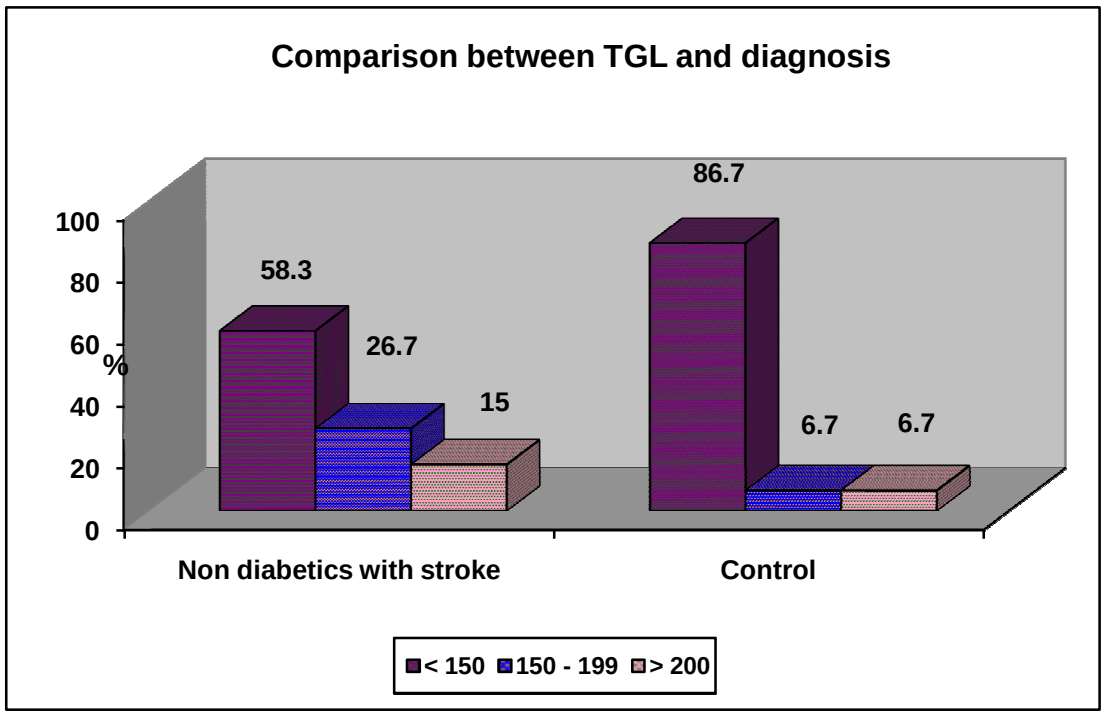
Total Cholesterol	Non diabetics with stroke	Control
< 200	43.3	90
200 – 240	16.7	5
> 240	40	5



43.3% of non-diabetics with stroke has total cholesterol less than 200. 16.7% of the same group had cholesterol 200-240. 40% of the same group has cholesterol more than 240%. Maximum patients 43.3 % has normal levels of total cholesterol. In controls, 90% have normal cholesterol values less than 200. 5% has cholesterol 200-240. 5% has cholesterol more than 240.

TRIGLYCERIDES X DIAGNOSIS

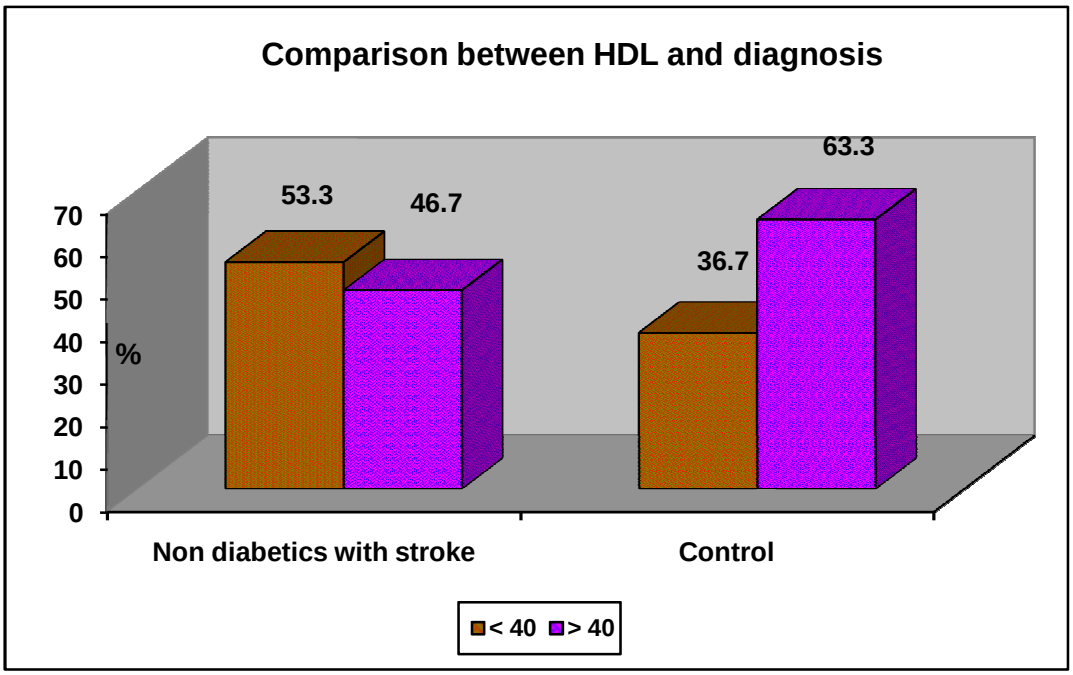
Triglycerides	Non Diabetics with Stroke	Control
< 150	58.3	86.7
150 – 199	26.7	6.7
> 200	15	6.7



In non-diabetics with stroke, less than 150 triglyceride value was 58.3%; 150-199 was 26.7%. > 200 was 15%. Maximum patients, 58.3% has normal tri-glycerides.In controls, 86% has triglyceride<150, 6% has 150-199%, in 6% more than 200. Maximum controls 86% has normal triglyceride values.

HDL X DIAGNOSIS

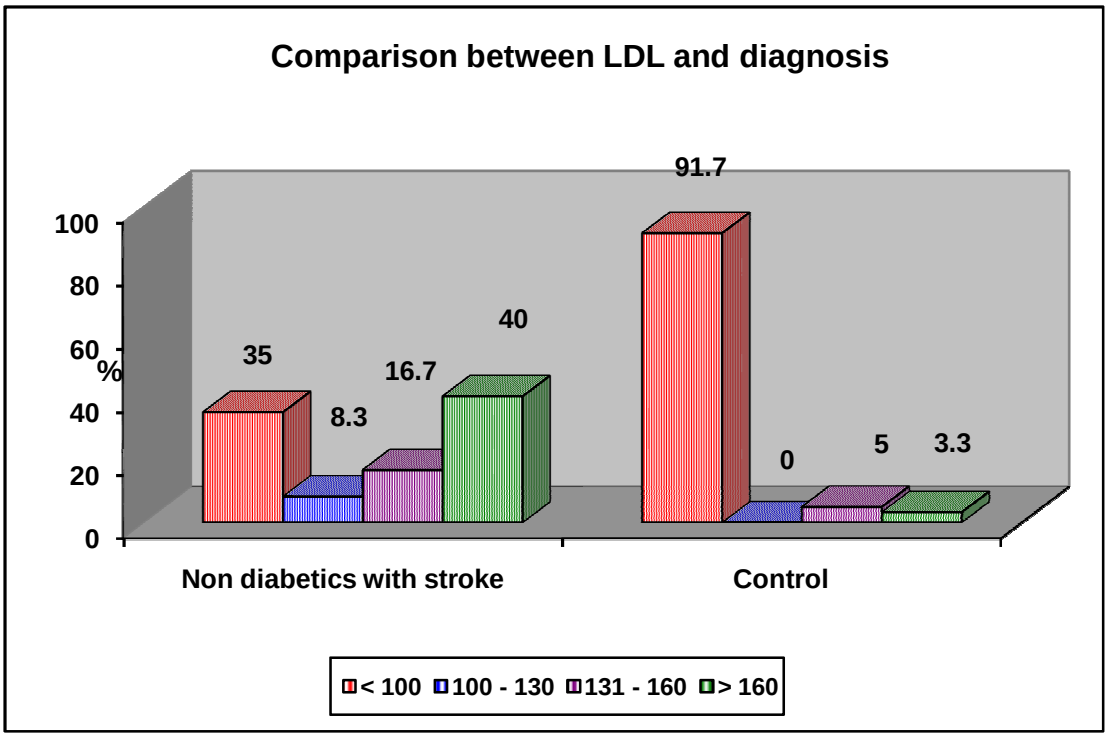
HDL	Non diabetics with stroke	Control
< 40	53.3	36.7
> 40	46.7	63.3



In non-diabetics with stroke, 53.3% had Hdl cholesterol < 40. And 46.7 % had Hdl cholesterol >40%. Maximum number of patients 53.3% had low HDL cholesterol. In control group, 36.7% had HDL < 40. 63.3% had HDL>40%. Maximum number of controls 63.3% had normal HDL values.

LDL X DIAGNOSIS

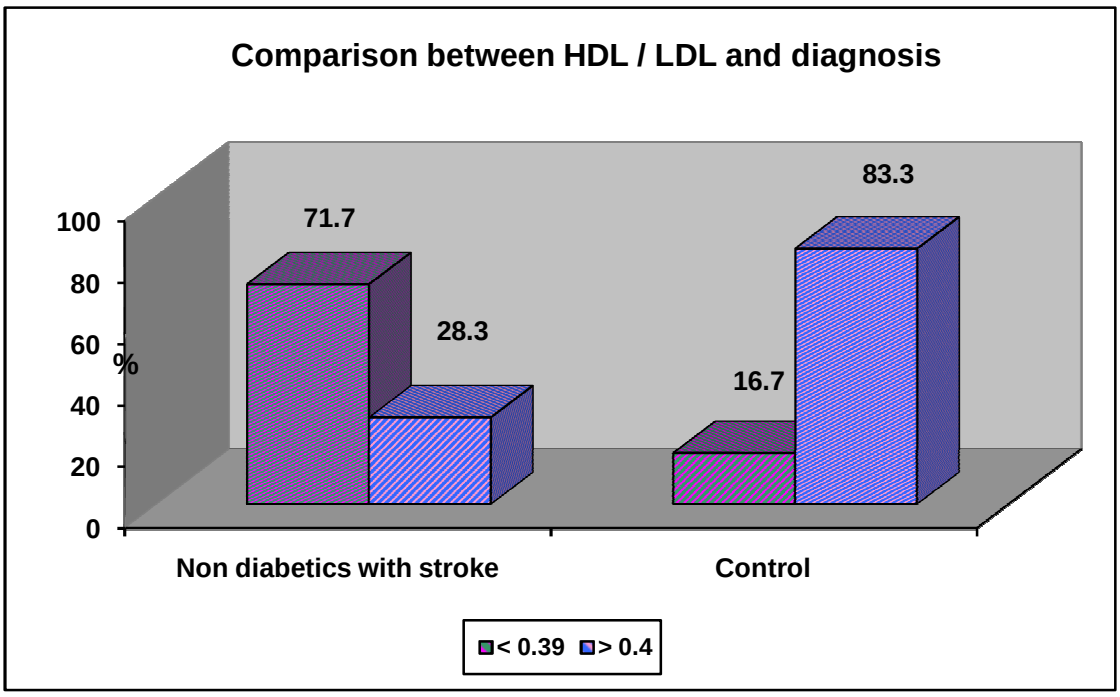
LDL	Non diabetics with stroke	Control
< 100	35	91.7
100 – 130	8.3	0
131 – 160	16.7	5
> 160	40	3.3



In non-diabetics with stroke, 35% had LDL<100, 8.3% had LDL 100-130, 15.7% had LDL 131-160, 40% had LDL > 160. Maximum number of patients 40% had LDL >160. In controls, 91.7 % has normal LDL values< 100. 5% had LDL 131-160. 8.3% had LDL >160.

HDL / LDL X DIAGNOSIS

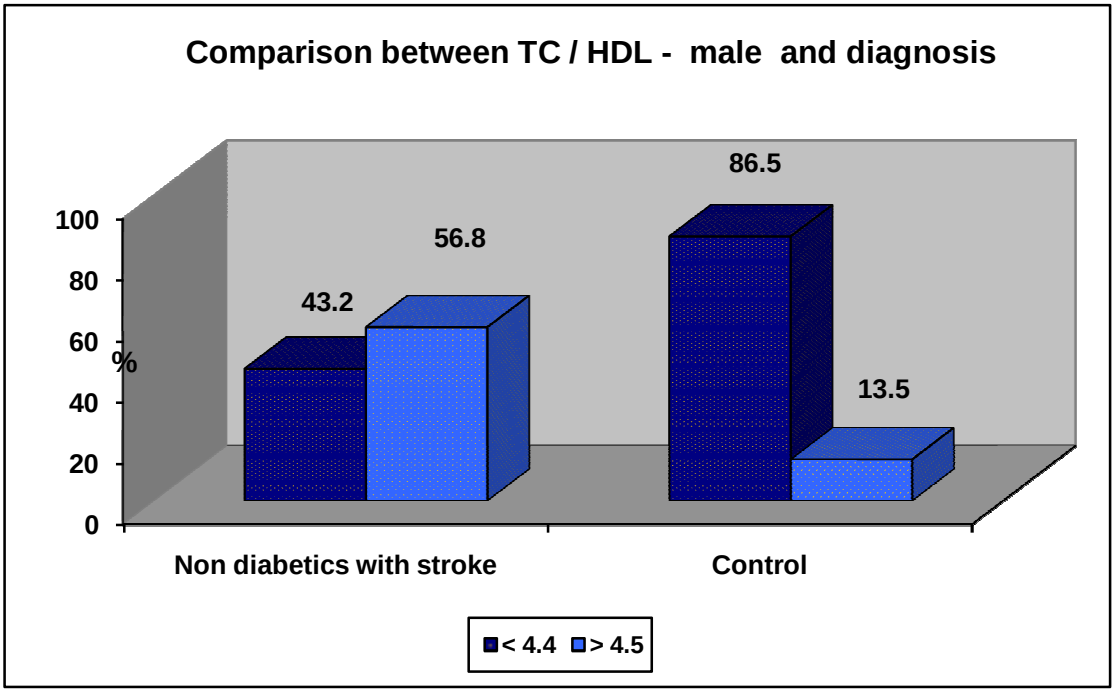
HDL/LDL	Non diabetics with stroke	Control
< 0.39	71.7	16.7
≥ 0.4	28.3	83.3



In non-diabetics with stroke, 71.7 % had HDL/LDL ratio<0.39. only 28.3% of the same group had HDL/LDL ratio>0.4%. In control group, 83.3% had HDl/LDL ratio >0.4. and 16.7% had HDL/LDL ratio>0.4.

TC / HDL X DIAGNOSIS – MALE

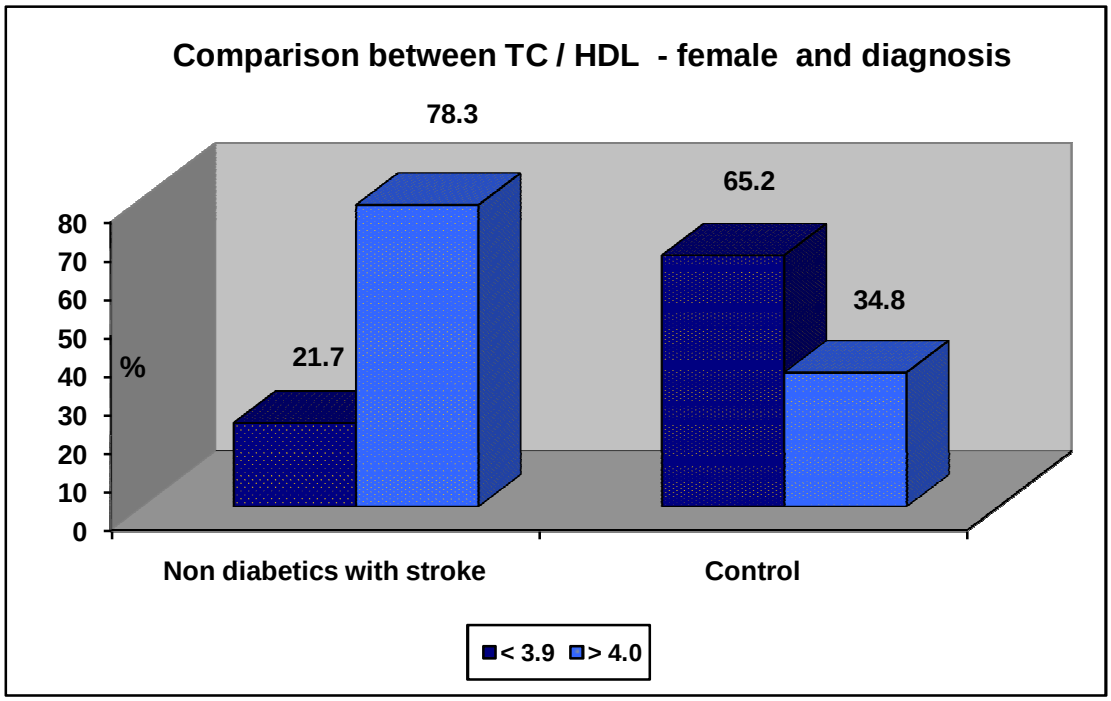
TC/HDL	Non diabetics with stroke	Control
< 4.4	43.2	86.5
> 4.5	56.8	13.5



In non-diabetic stroke males, 56.8% has TC/HDL >4.5 and 43.2% has <4.4. maximum number of patients had TC/HDL ratio. In control group, 86.5% has TC/HDL ratio in males <4.4 and 13.5% had > 4.5

TC / HDL X DIAGNOSIS - FEMALE

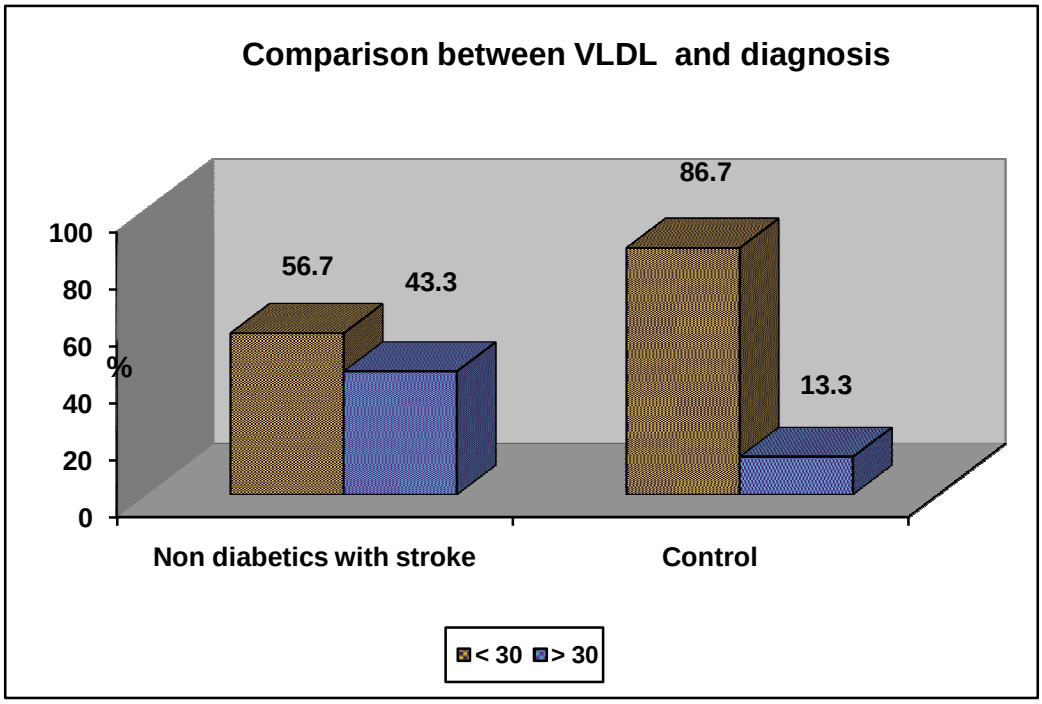
TC/HDL	Non diabetics with stroke	Control
< 3.9	21.7	65.2
> 4.0	78.3	34.8



In non-daibetic stroke with females, maximum number had 78.3% has TC/HDL ratio >4. And 21.7% had ratio less than 3.9%. in control group, maximum number of controls, 65.2% has TC/HDL < 3.9. 35.8% has TC/HDL more than 4.

VLDL X DIAGNOSIS

VLDL	Non diabetics with stroke	Control
< 30	56.7	86.7
> 30	43.3	13.3



In non-diabetics with stroke, 56.7% had VLDL< 30. 43.3% had VLDL >30%. Maximum number of patients has VLDL normal values. 86.7% of controls had normal VLDL <30%. 13.3% of controls have high VLDL values >30.

DISCUSSION

Association of total Cholesterol to Non diabetics with stroke

In our study conducted on 60 patients showed total cholesterol was elevated in non- diabetics with stroke compared to the control group was highly significant P<0.001.

A study on lipid profile in non-diabetics with stroke done by Sreedharan-[2] et al in 2010 showed a definite increase in serum total cholesterol in non- diabetic stroke patients when compared to control groups. In his study he showed both ischemic and hemorrhagic stroke are associated with increased cholesterol levels.

Benfante et al-[17] (stroke 1994) showed elevated serum cholesterol is a risk factor for both coronary heart disease and thromboembolic stroke in Hawaiian Japanese Men.

Di Mascio et al showed a positive association between risk of stroke and serum cholesterol.

Iso-[31] et al emphasised an inverse association between serum cholesterol level and hemorrhagic stroke but in his study there was a positive association with non – hemorrhagic stroke.

Tanizaki et al had showed total cholesterol was an addition risk factor for cardio embolic stroke in females.

Linden strorn et al in 1994 showed low total cholesterol levels decrease stroke in coronary artery disease patient.

Alok Mohankar et al 1993 observed increase total cholesterol lead to increased incidence of atherosclerosis of large vessels. Atherosclerosis is a definite risk factor for stroke.

Triglycerides association with non – diabetic stroke

The serum triglycerides were high in our patients compared to the control group of our study showing statistical significance ($P<0.05$)

Sreedharan-[2] et al in his study showed 80% of non-diabetic stroke patients with S.triglyceride >200mg/dl had ischemic stroke and the remaining 20% had hemorrhagic stroke.

Tilvis R.S.-[4] et al in his study had showed serum triglyceride is higher in ischemic stroke. Farid et al also had similar results in his study in 1972.

Albucher J.K-[12] et al 2000 has showed serum triglycerides in normal range in his study on stroke.

Hachinski-[20] et al showed a positive association of triglycerides in patients of atherothrombotic stroke and transient ischemic attacks.

Association of serum HDL Cholesterol

The levels of serum HDL cholesterol is not significant in this study conducted on 60 non-diabetic stroke patients.

Simons et al study revealed HDL cholesterol had protective effect on ischemic stroke.

The northern Manhattan study on stroke in 2001, concluded higher values of HDL cholesterol was associated with reduced risk of stroke.

Alok Mohankar-[3], Ravindrakumar in 1993 showed increased LDL levels and low HDL levels were associated with atherosclerosis.

Wanna Mithee S.G et al had shown high HDL levels were associated with decreased non fatal stroke risk.

A study by Rubens et al in 2001 showed Gemfibrosil which raises HDL – cholesterol level decrease ischemic stroke by 31% in men.

Albucher-[12] et al study clearly indicated HDL – Cholesterol as the only lipid associated with stroke risk. He emphasised the need for management of low HDL cholesterol in young patients regardless of atherosclerosis.

Association of serum LDL Cholesterol

The levels of serum LDL cholesterol was highly significant in our study conducted on 60 non – diabetics with stroke ($P < 0.001$)

Sreedharan -[2]et al showed raised levels of serum LDL cholesterol had significant risk of ischemic stroke in non-diabetics.

Bolet et al and Hachinski-[8] et al have showed positive correlation between LDL cholesterol levels and risk of stroke.

Anseil B.J et al in 2000 showed patients with established atherosclerosis showed are treated with statins to lower LDL cholesterol levels < 100 mg to decrease the incidence of stroke.

Kurth T-[6] et al 2007 showed remarkable increase in serum LDL levels in ischemic stroke patients.

VLDL :-

VLDL levels were significantly elevated in our study conducted on 60 non-diabetics with stroke and control group.

Bidyadhar-[7] et al 1984 showed that VLDL was raised in their study on stroke.

Sreedharan -[2]et al in his study showed high VLDL was not associated with risk of stroke in non-diabetic patients.

CONCLUSION

Our study was conducted on 60 non-diabetic stroke patients and 60 controls. Exclusion was done because diabetes is associated with hyperlipidemia and atherosclerosis.

This study showed significant association of total cholesterol, triglycerides, LDL cholesterol in non-diabetics with stroke. High levels of total cholesterol, triglycerides, LDL cholesterol are associated with higher risk of stroke.

Lowered HDL cholesterol levels were not significantly associated with stroke. The ratio of HDL/LDL Cholesterol, TG/HDL cholesterol was calculated.

Dyslipidemia is a tip in iceberg. Dyslipidemia if properly treated being a modifiable risk factor for stroke it decreasing the incidence of stroke due to dyslipidemia. This leads to decreased morbidity and mortality leading to a healthier society.

LIMITATIONS

A prospective study of non-diabetic stroke patients with follow up would have been of greater value to draw more definitive conclusion.

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ANNEXURES

ABBREVIATIONS

1. CVA- Cerebro vascular accident
2. TIA- Transient ischemic attack
3. WHO- World health organisation
4. RIND- Reversible ischemic neurological deficit
5. LDL- Low density lipo-protein
6. HDL- High density lipo-protein
7. VLDL- Very low density lipo-protein
8. IDL- Intermediate density lipo-protein
9. LCAT- Lecithin cholesterol acyl transferase
10. T.Cholesterol- Total cholesterol
11. HMG CoA- 3-Hydroxy 3-Methyl glutarate co-enzyme A
12. ANA- Anti-Nuclear antibodies
13. MCA- Middle cerebral artery
14. LPL- Lipo protein lipase

PROFORMA

h/o stroke

duration:

NAME: UNIT NO.:

I.P/O.P.NO.:

AGE/SEX:

OCCUPATION: DATE:

ADDRESS:

CONTACT NO:

COMPLAINTS:

HISTORY:

PAST HISTORY:

Hypertension/ Dyslipidemia/ CAD/ CKD

FAMILY HISTORY:Dyslipidemia

PERSONAL HISTORY: Chewing Tobacco/ Smoking/ Alcohol/ Drug Abuse

TREATMENT HISTRY(DRUGS):

GENERAL PHYSICAL EXAMINATION:

HT: WT: BMI:

VITALS:

BP: PR: RR:

SYSTEMIC EXAMINATION:

CVS:

RS:

P/A:

CNS:

Others

INVESTIGATIONS:

Complete bood count:

RFT

Random blood sugar	Urea	creatininie

LIPID PROFILE

Triglycerides	Total Cholesterol	HDL- C	LDL-C	VLDL-C

IMAGING STUDIES;

ECG

ECHO

CT/MR

ISIGNATURE OF INVESTIGATOR SIGNATURE OF GUIDE

MASTER CHART

S.No.	Name	Age	Sex	Diagnosis	Ht	Wt	BMI	Smoking	Hyper tension	T.cho	T.gly	HDL	LDL	VLDL	HDL | LDL	TC | HDC
1	RAMACHANDRAN	60	M	Non Diabetics with stroke	160	60	23.43	-	-	271	146	60	181.8	29.2	0.33	4.51
2	RAMAN	64	M	Non Diabetics with stroke	158	65	26.03	+	+	247	132	42	178.6	26.4	0.23	5.88
3	SYED JALAL	73	M	Non Diabetics with stroke	170	84	29.06	-	-	126	134	28	71.2	26.8	0.39	4.5
4	VIJAYAGOPAL	56	M	Non Diabetics with stroke	164	74	27.5	+	-	212	119	48	140.2	23.8	0.34	4.41
5	GOVINDASAMY	51	M	Non Diabetics with stroke	172	68	22.98	-	-	267	156	36	199.8	31.2	0.18	7.41
6	KUPPUSAMY	50	M	Non Diabetics with stroke	160	64	25	-	-	184	133	46	111.4	26.6	0.41	4
7	HAKKIM	41	M	Non Diabetics with stroke	168	76	26.92	-	-	280	158	34	214.4	31.6	0.15	8.23
8	BABU	36	M	Non Diabetics with stroke	155	64	23.5	+	+	146	112	54	69.6	22.4	0.77	2.7
9	RAJAGURU	32	M	Non Diabetics with stroke	163	73	27.47	-	-	257	163	32	192.4	32.6	0.16	8
10	VENUGOPAL	80	M	Non Diabetics with stroke	161	62	23.91	+	+	254	136	53	173.8	27.2	0.29	4.7
11	BABU	35	M	Non Diabetics with stroke	154	66	27.82	+	-	257	171	62	160.8	34.2	0.38	4.14

12	SANTHANAM	56	M	Non Diabetics with stroke	174	66	21.8	-	+	124	112	31	70.6	22.4	0.43	4
13	SALEEM	36	M	Non Diabetics with stroke	168	74	26.21	+	+	253	227	56	151.6	45.4	0.36	4.51
14	SENTHIL MURUGAN	36	M	Non Diabetics with stroke	156	84	34.51	-	+	100	110	28	50	22	0.56	3.57
15	SUBRAMANIYAN	58	M	Non Diabetics with stroke	162	56	21.33	-	-	232	164	51	148.2	32.8	0.34	4.54
16	RAYAPPAN	60	M	Non Diabetics with stroke	158	68	27.23	-	-	248	122	33	190.6	24.8	0.17	7.51
17	KAMARAJ	54	M	Non Diabetics with stroke	154	60	25.29	+	-	168	136	58	82.8	27.2	0.69	2.89
18	DURAI	45	M	Non Diabetics with stroke	168	64	22.67	-	-	138	107	36	80.6	21.4	0.44	3.83
19	KIRUBAKARAN	75	M	Non Diabetics with stroke	159	67	26.5	-	-	261	168	63	164.4	33.6	0.38	4.14
20	MATHIVANAN	54	M	Non Diabetics with stroke	163	73	27.47	+	+	120	133	60	33.4	26.6	1.81	2
21	KENGAN	65	M	Non Diabetics with stroke	171	68	23.25	+	-	251	181	43	171.8	36.2	0.3	5.83
22	NARAYANAN	58	M	Non Diabetics with stroke	176	62	20.01	-	-	243	214	37	163.2	42.8	0.22	6.56
23	RAJENDRAN	65	M	Non Diabetics with stroke	152	58	25.1	+	+	108	107	47	39.6	21.4	1.175	2.29
24	MURUGAN	46	M	Non Diabetics with stroke	163	63	23.71	+	+	244	148	34	180.4	29.6	0.18	7.17

25	MUNUSAMY	67	M	Non Diabetics with stroke	168	59	20.9	-	-	103	124	51	27.2	24.8	1.875	2.019
26	SEKAR	50	M	Non Diabetics with stroke	148	55	25.11	+	-	242	174	29	178.2	34.8	0.162	8.34
27	SHANMUGAM	62	M	Non Diabetics with stroke	157	52	21.09	-	+	224	136	53	143.8	27.2	0.36	4.22
28	RANGANATHAN	78	M	Non Diabetics with stroke	154	50	21.08	-	-	148	96	50	78.8	19.2	0.63	2.96
29	NITHYA NANDHAN	41	M	Non Diabetics with stroke	159	58	22.9	+	-	107	129	33	48.2	25.8	0.68	3.2
30	GOPALAN	95	M	Non Diabetics with stroke	158	62	24.83	-	-	251	146	47	174.8	29.2	0.268	5.3
31	MUNUSAMY	62	M	Non Diabetics with stroke	163	61	22.96	-	+	304	204	42	221.2	40.8	0.19	7.2
32	SIVARAMAN	48	M	Non Diabetics with stroke	168	66	23.38	-	-	168	132	27	114.6	26.4	0.23	6.2
33	RAJU	57	M	Non Diabetics with stroke	162	72	27.43	-	-	272	230	43	183	46	0.23	6.32
34	CHINNAPPAN	64	M	Non Diabetics with stroke	155	58	24.14	-	-	156	121	34	100.8	24.2	0.3	5.03
35	NAGARAJAN	59	M	Non Diabetics with stroke	153	57	24.35	-	+	220	153	46	143.4	30.6	0.32	4.7
36	RAVI	66	M	Non Diabetics with stroke	156	63	25.88	-	-	232	204	37	154.2	40.8	0.23	6.27
37	MUNUSAMY	83	M	Non Diabetics with stroke	164	63	23.42	+	-	135	116	51	60.8	23.2	0.83	2.6

38	PRABAVATHY	50	F	Non Diabetics with stroke	157	54	21.9	-	-	248	107	29	197.6	21.4	0.14	8.5
39	JAIRUBA	63	F	Non Diabetics with stroke	158	60	24.03	-	-	246	158	50	164.4	31.6	0.3	4.92
40	PONNUMANI	58	F	Non Diabetics with stroke	155	58	24.18	-	-	101	96	32	49.8	19.2	0.64	3.15
41	VIMALA	68	F	Non Diabetics with stroke	156	71	29.17	-	-	218	163	48	138	32.6	0.34	4.54
42	HEMALATHA	58	F	Non Diabetics with stroke	155	58	24.14	-	+	170	108	37	112	21.6	0.33	4.59
43	SHOBA	46	F	Non Diabetics with stroke	157	63	25.55	-	-	247	174	45	167.2	34.8	0.26	5.48
44	VIDHYA	59	F	Non Diabetics with stroke	148	55	25.11	-	-	144	102	34	89.6	20.4	0.37	4.23
45	ANDAL	57	F	Non Diabetics with stroke	162	72	27.43	-	+	216	182	39	140.6	36.4	0.27	5.53
46	ROJA	67	F	Non Diabetics with stroke	163	70	26.34	-	-	103	112	43	37.6	22.4	1.13	2.39
47	PREMA	58	F	Non Diabetics with stroke	160	72	28.12	-	-	159	142	36	94.6	28.4	0.37	4.41
48	PANCHALI	70	F	Non Diabetics with stroke	161	62	23.91	-	-	212	155	41	140	31	0.29	5.1
49	JAYANTHI	67	F	Non Diabetics with stroke	154	66	27.82	-	-	283	306	28	193.8	61.2	0.14	10.1
50	RAVANAMME	64	F	Non Diabetics with stroke	158	68	27.23	-	-	127	102	47	59.6	20.4	0.78	2.7

51	RANGANAYAKI	64	F	Non Diabetics with stroke	151	61	26.75	-	-	147	104	27	99.2	20.8	0.27	5.44
52	SUMATHY	62	F	Non Diabetics with stroke	147	59	27.3	-	-	256	204	34	181.2	40.8	0.18	7.52
53	RUKMANI	54	F	Non Diabetics with stroke	153	68	29.05	-	-	143	108	26	95.4	21.6	0.27	5.5
54	MAHESWARI	60	F	Non Diabetics with stroke	157	46	18.66	-	-	250	160	28	190	32	0.14	8.92
55	BANU	52	F	Non Diabetics with stroke	150	40	17.76	-	-	234	168	33	168	33.6	0.19	7.09
56	LEELAVATHY	75	F	Non Diabetics with stroke	149	58	26.12	-	-	107	96	37	51	19.2	0.72	2.89
57	RADHA	68	F	Non Diabetics with stroke	154	62	26.14	-	-	168	132	31	111	264	0.27	5.41
58	RAGANAMMA	62	F	Non Diabetics with stroke	151	44	19.3	-	-	230	206	36	153	41.2	0.23	6.38
59	KALPANA	61	F	Non Diabetics with stroke	148	61	27.85	-	-	104	98	35	50	19.6	0.7	2.97
60	BAGAVATHI	56	F	Non Diabetics with stroke	156	60	24.65	-	-	258	208	28	189	41.6	0.14	9.21
61	PALANI	57	M	CONTROL	154	58	24.46	-	-	166	134	48	91.2	26.8	0.52	3.45
62	AMUDHAN	65	M	CONTROL	150	60	26.67	-	-	132	120	34	74	24	0.45	3.88
63	ANANDHAN	44	M	CONTROL	156	64	26.3	-	-	162	132	42	93.6	26.4	0.44	3.85

64	VELU	74	M	CONTROL	160	68	26.5	-	-	148	121	44	79.8	24.2	0.55	3.36
65	MARIMUTHU	80	M	CONTROL	148	54	24.65	-	-	151	123	43	83.4	24.6	0.51	3.51
66	RAJAGURU	60	M	CONTROL	157	66	26.78	-	-	131	156	36	63.8	31.2	0.56	3.04
67	RAMESH	52	M	CONTROL	162	70	26.67	-	-	243	162	53	157.6	32.4	0.33	4.58
68	PRAKASH	37	M	CONTROL	158	60	24.03	-	-	128	124	44	59.2	24.8	0.74	2.9
69	VELAN	50	M	CONTROL	150	65	28.89	-	-	134	121	43	66.8	24.2	0.643	3.11
70	MURUGAN	34	M	CONTROL	162	54	20.58	-	-	124	126	50	48.8	25.2	1.02	2.48
71	MANOHAR	37	M	CONTROL	156	62	25.48	-	-	156	131	45	84.8	26.2	0.53	3.46
72	MADHAVAN	59	M	CONTROL	154	60	25.3	-	-	138	125	38	75	25	0.5	3.63
73	CHINNAPPAN	55	M	CONTROL	157	64	25.96	-	-	140	123	51	64.4	24.6	0.791	2.745
74	SELVAKUMAR	58	M	CONTROL	160	70	27.34	-	-	246	204	47	158.2	40.8	0.297	5.23
75	SURYA	34	M	CONTROL	162	64	24.39	-	-	142	127	53	63.6	25.4	0.83	2.67
76	VIMALAN	46	M	CONTROL	163	58	21.83	-	-	148	128	43	79.4	25.6	0.541	3.44

77	VIJAYAKUMAR	37	M	CONTROL	158	60	24.03	-	-	140	130	34	80	26	0.425	4.11
78	VAIDHIYANADHAN	76	M	CONTROL	151	54	23.68	-	-	156	129	49	81.2	25.8	0.603	3.18
79	SRIDHAR	60	M	CONTROL	168	60	21.26	-	-	143	133	44	72.4	26.6	0.607	3.25
80	PRABAKARAN	55	M	CONTROL	160	54	21.09	-	-	151	120	36	91	24	0.395	4.19
81	PUNYAKODI	59	M	CONTROL	154	64	26.99	-	-	137	204	50	46.2	40.8	1.08	2.74
82	DHARMAN	66	M	CONTROL	159	63	24.92	-	-	143	121	37	81.8	24.2	0.45	3.86
83	DEIVEGAN	48	M	CONTROL	160	57	22.27	-	-	150	131	44	69.8	26.2	0.63	3.4
84	MANIVANNAN	66	M	CONTROL	162	70	26.67	-	-	238	126	50	162.8	25.2	0.3	4.76
85	VASAN	69	M	CONTROL	161	58	22.38	-	-	151	134	43	81.2	26.8	0.52	3.51
86	VAIRAM	63	M	CONTROL	158	62	24.84	-	-	154	125	33	96	25	0.34	4.66
87	SARAVANAN	50	M	CONTROL	152	57	24.67	-	-	140	127	41	73.6	25.4	0.55	3.41
88	SHANMUGAN	75	M	CONTROL	168	60	21.26	-	-	138	130	51	61	26	0.83	2.7
89	SRIDHAR	43	M	CONTROL	161	63	24.3	-	-	143	128	48	69.4	25.5	0.69	2.97

90	VELAN	91	M	CONTROL	163	68	25.59	-	-	151	133	46	78.6	26.6	0.58	3.28
91	THANIKACHALAM	63	M	CONTROL	162	66	25.15	-	-	138	131	43	68.8	26.2	0.625	3.2
92	MAGESWARAN	46	M	CONTROL	156	54	22.19	-	-	142	120	33	85	24	0.38	4.3
93	DIVAKARAN	55	M	CONTROL	153	58	24.78	-	-	126	122	51	50.6	24.4	1.007	2.47
94	SIVALINGAM	66	M	CONTROL	158	62	24.84	-	-	212	131	36	49.8	26.2	0.24	5.8
95	THENNAPPAN	57	M	CONTROL	163	67	25.22	-	-	131	124	45	61.2	24.8	0.735	2.9
96	BALU	64	M	CONTROL	160	70	27.34	-	-	128	123	39	64.4	24.6	0.6	3.28
97	KIRUBAKARAN	81	M	CONTROL	158	60	24.03	-	-	137	126	34	77.8	25.2	0.43	4.02
98	DHANABAKYAM	55	F	CONTROL	152	51	22.07	-	-	142	120	41	77	24	0.53	3.46
99	ESAIVANI	50	F	CONTROL	154	58	24.46	-	-	124	204	43	40.2	40.8	1.06	2.88
100	SARASWATHY	61	F	CONTROL	160	64	25	-	-	128	131	35	66.8	26.2	0.52	3.65
101	VASUKI	67	F	CONTROL	147	55	25.45	-	-	138	160	46	60	32	0.76	3
102	MARIYAMMA	55	F	CONTROL	154	60	25.3	-	-	142	121	35	82.8	24.2	0.42	4.05

103	RANI	43	F	CONTROL	158	62	24.84	-	-	121	133	44	50.4	26.6	0.87	2.75
104	PARVATHI	55	F	CONTROL	155	50	20.81	-	-	206	158	34	140.4	31.6	0.24	6.05
105	PECHAIAMMA	52	F	CONTROL	153	55	23.5	-	-	137	126	33	78.8	25.2	0.41	4.151
106	ELIZABETH	53	F	CONTROL	160	64	25	-	-	150	202	58	51.6	40.4	1.12	2.586
107	SARADHA	63	F	CONTROL	157	54	21.91	-	-	149	124	31	93.2	24.8	0.33	4.806
108	NIRMALA	65	F	CONTROL	159	63	24.92	-	-	242	131	53	162.8	26.2	0.32	4.56
109	NARASAMMA	70	F	CONTROL	152	49	21.21	-	-	146	133	39	80.4	26.6	0.48	3.74
110	DIVYA	63	F	CONTROL	151	55	24.12	-	-	151	131	51	73.8	26.2	0.69	2.96
111	SATHYA	62	F	CONTROL	154	56	23.61	-	-	144	121	54	65.8	24.2	0.82	2.6
112	RADHA	63	F	CONTROL	158	60	24.03	-	-	151	124	32	94.2	24.8	0.33	4.71
113	SUJATHA	51	F	CONTROL	157	61	24.75	-	-	153	123	51	77.4	24.6	0.65	3
114	SUDHA	53	F	CONTROL	151	53	23.24	-	-	147	131	35	85.8	26.2	0.4	4.2
115	SAVITHRI	60	F	CONTROL	148	52	23.74	-	-	138	120	53	61	24	0.86	2.6

116	MADHAVI	65	F	CONTROL	150	51	22.67	-	-	133	122	34	74.6	24.4	0.45	3.91
117	DEVI	73	F	CONTROL	153	54	23.07	-	-	156	131	38	91.8	26.2	0.41	4.1
118	DHANAMMA	66	F	CONTROL	151	55	24.12	-	-	148	133	45	76.4	26.6	0.58	3.28
119	SRIDEVI	62	F	CONTROL	152	56	24.24	-	-	135	128	36	73.4	25.6	0.49	3.75
120	SUBA	53	F	CONTROL	155	53	22.06	-	-	151	132	44	80.6	26.4	0.54	3.43

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.1589/ME-1/Ethics/2014 Dt:06.03.2014.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study of lipid profile in Non Diabetics with stroke" – For Project Work submitted by Dr.S.Mythili, MD (GM), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

29/5/14
CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

GOVT. KILPAUK MEDICAL COLLEGE * Chennai-10. * 29 MAY 2014